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# LOSS OF AXIS-CYLINDERS IN SCLEROTIC PLAQUES AND SIMILAR LESIONS

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CHARCOT [1] was apparently the first to point out the essential histologic features of sclerotic plaques: the absence of myelin, the presence of axis-cylinders, the fibrous gliosis and the perivascular infiltration with phagocytic cells. This much of his description has been copied in almost every textbook. Two of the other alterations which he clearly recognized have since appeared and disappeared repeatedly in scientific literature: the narrowing and closure of vessels and the loss of axis-cylinders, which, he wrote, "persist in certain number."

Chiefly, apparently, on the basis of the histology of sclerotic plaques Charcot founded the important distinction between secondary and primary degeneration of myelin. Secondary degeneration is the result of the destruction of axis-cylinders, or nerve cells. The neurofibrils disappear distal to the lesion, and the myelin then begins to disintegrate. Primary degeneration is a process which attacks almost all the myelin sheaths in an area of tissue, leaving some or many axis-cylinders intact and giving rise to no conspicuous secondary degeneration. This is a formal definition; in practice a pure primary degeneration is rare or nonexistent. Usually a few axis-cylinders are destroyed, as in the cases Charcot described, and their fibrils degenerate distally but are so scattered that they are not easy to demonstrate. Mere damage to an axis-cylinder, such as may occur in "demyelinating" lesions, does not cause a descending degeneration.

Since the advent of the Weigert stain, which demonstrates loss of myelin so strikingly, the attention of neurologists has tended to become

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1. Charcot, J. M.: Histologie de la sclérose en plaques, Gaz. d. hôp. **41**:554-555, 557-558 and 566, 1868.

centered on the myelin lesion, both in primary and in secondary degeneration. As fixation and embedding for the Weigert process usually make a study of other tissue elements difficult, there has been a tendency to neglect these structures. This has been particularly true of the axis-cylinders.

## PREVIOUS STUDIES OF THE AXIS-CYLINDERS IN SCLEROTIC PLAQUES

A large number of studies have, however, been directed toward the axis-cylinders. Those which were made before the introduction of the Bielschowsky technic may be disregarded (with the exception perhaps of Charcot's, already mentioned, and a few others). The results of these investigations up to 1935 have been well summarized by Marburg.[2] From an extensive review of the literature and his own numerous observations, he drew the conclusion that the axis-cylinders are usually damaged and in many instances completely destroyed. This is clearly shown in two of his illustrations, of sections stained with the Bielschowsky technic.

While many pathologists have reported damage to axis-cylinders in the plaques of multiple sclerosis, there seems to be a widespread opinion that the extent of damage is relatively slight. Spielmeyer[3] spoke of "the characteristic persistence of axis-cylinders, which are noted in the majority of lesions and in the majority of cases of multiple sclerosis in great predominance, an observation which is surprising each time, and which constitutes the essential peculiarity of the disease."

There have been two recent contributions to the problem. In an extensive survey of lesions of multiple sclerosis,[4] numerous plaques were encountered, especially in the white matter of the hemispheres, in which there appeared to be an extensive loss of axis-cylinders, of possible functional significance. The observations were made chiefly on thin sections stained with the Mallory, Masson and similar technics, but similar observations were made on material stained by the Davenport silver method. Among the lesions studied, there were many in which only sparse axis-cylinders were seen.

These results were criticized by Greenfield and King[5] on the ground that the stains employed were not sufficiently specific for axis-cylinders. Using the original Bielschowsky technic, of course on frozen sections, they examined over 125 cerebral plaques from 13 cases. They reported:

2. Marburg, O.: Multiple Sklerose, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 13, pp. 546-693.

3. Spielmeyer, W.: Infektion und Nervensystem, Deutsche Ztschr. f. Nervenh. **110**:290-295, 1929.

4. Putnam, T. J.: Studies in Multiple Sclerosis: VIII. Etiologic Factors in Multiple Sclerosis, Ann. Int. Med. **9**:854-863 (Jan.) 1936.

5. Greenfield, J. G., and King, L. S.: Observations on the Histopathology of the Cerebral Lesions in Disseminated Sclerosis, Brain **59**:445-458 (Dec.) 1936.

Less than 10 per cent showed what could be called "severe destruction," the remainder being fairly evenly divided into slight or no loss, and moderate diminution. The extreme degree was not only rare, but where present was always in tissue that was highly rarefied or spongy. [This description fits well the plaques in which gliosis is slight, such as are common in the white matter of the hemispheres.]

What we call severe destruction is reduction in number to one-fifth or one-seventh (approximately) of the normal density of nerve fibers.

They reported no fiber counts, however. In two plaques of 1 case there was extreme softening, so that they could not be cut in frozen section except with gelatin embedding. These two plaques were left out of the statistical study. The authors give no illustrations of axis-cylinders to demonstrate their point.

To a certain extent the criticism of Greenfield and King is justified. There can be no doubt that the Bielschowsky method stains some axis-cylinders which may be missed with anilin dye stains, and even with many of the older silver stains on embedded tissue.

The Bielschowsky stain has its limitation, however. It depends on frozen sections, which are almost impossible to cut from large plaques of intense degeneration, where axis-cylinders are fewest, as the experience of Greenfield and King shows. Frozen sections are necessarily many times as thick as the best paraffin sections, so that a moderate degree of loss of axis-cylinders is difficult to detect. Finally, the Bielschowsky technic is likely to stain a certain number of glial fibrils, especially in old fibrotic lesions of isomorphic gliosis. It has seemed worth while, therefore, to reopen the question.

The ideal method of resolving the difficulty would be to find a stain which combines the best features of the two technics which are found to give conflicting results. Fortunately, such a method has recently become available: the Bodian stain,[6] which depends on the reduction of a silver proteinate. It can be used on thin paraffin sections; the results are uniform, and there is a general agreement that it stains axis-cylinders with a unique completeness and selectivity. The clarity of the stain for low power enlargements is enhanced by incineration of the sections, as suggested by Rosett.[7]

## MATERIAL AND METHODS

Twenty-six blocks, containing 31 distinct plaques, were taken from embedded material from 7 cases of multiple sclerosis. The cases were unselected, except that 2 were included because they were particularly rich in acute lesions. One of these was of the neuromyelitis optica type. Sections cut at 5 microns were

6. Bodian, D.: A New Method for Staining Nerve Fibers and Nerve Endings in Mounted Paraffin Sections, Anat. Rec. **65**:89-97 (April) 1936; Staining of Paraffin Section of Nervous Tissues with Activated Protargol: Role of Fixatives, ibid. **69**:153-162 (Sept.) 1937.

7. Rosett, J.: A Method of Replacing Nerve Fibers with Fused Gold, Arch. Neurol. & Psychiat. **44**:442-443 (Aug.) 1940.

stained by the Bodian method, and adjacent sections, by the Smith-Quigley or the Mahon [8] method for myelin sheaths. Usually, a series of other stains was available from the same or from adjacent blocks.

For comparison with the lesions of multiple sclerosis, 9 blocks from 2 cases were studied in which there were numerous patches of partial degeneration resulting from such processes as arteriosclerosis and venous thrombosis. These blocks were embedded in paraffin, cut and stained as just described.

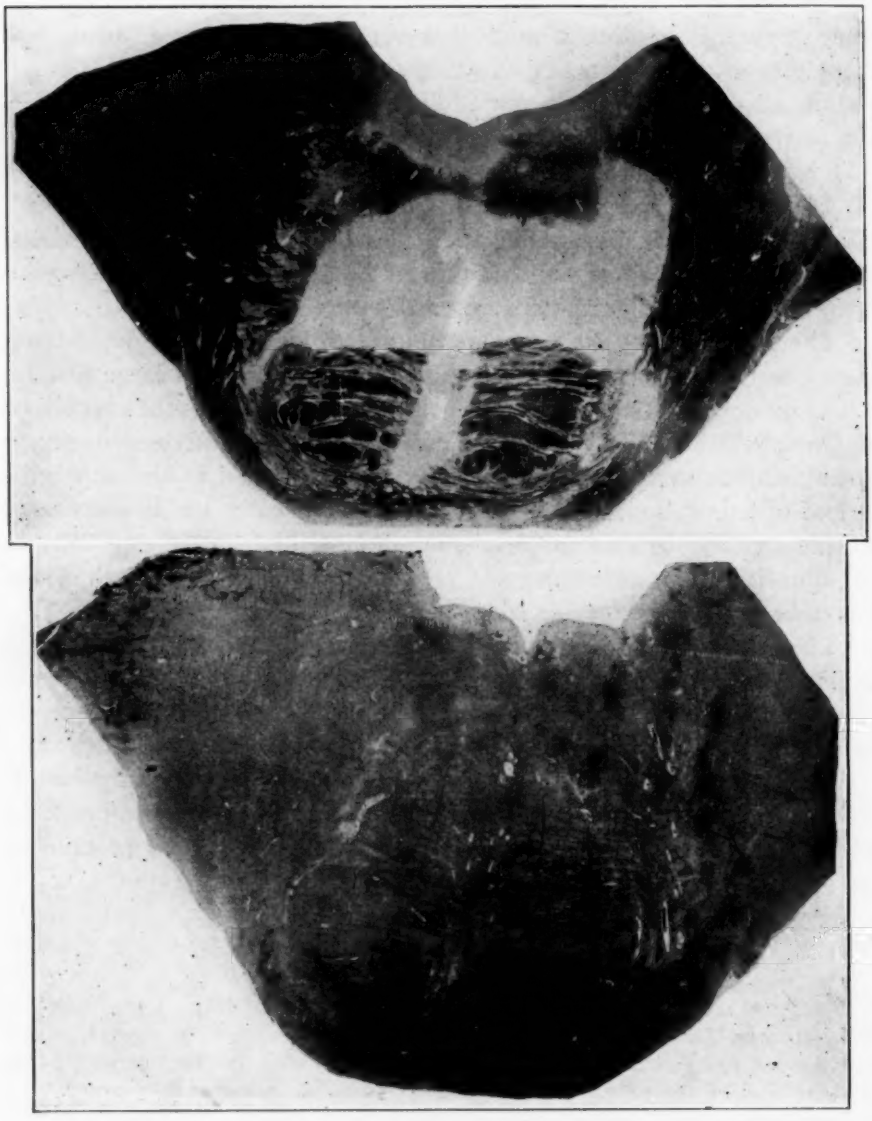

Fig. 1.—Sections from the same block of the pons from a case of multiple sclerosis. Above, Mahon stain for myelin; below, Bodian stain for axis-cylinders. Note apparent intactness of axis-cylinders. Lens enlargement.

8. Mahon, G. S.: Stain for Myelin Sheaths in Tissues Embedded in Paraffin, Arch. Neurol. & Psychiat. **38**:103-107 (July) 1937.

RESULTS

*Primary Degeneration.*—Of the 29 sclerotic plaques studied, all showed some degree of loss of axis-cylinders. In 2 blocks, both from

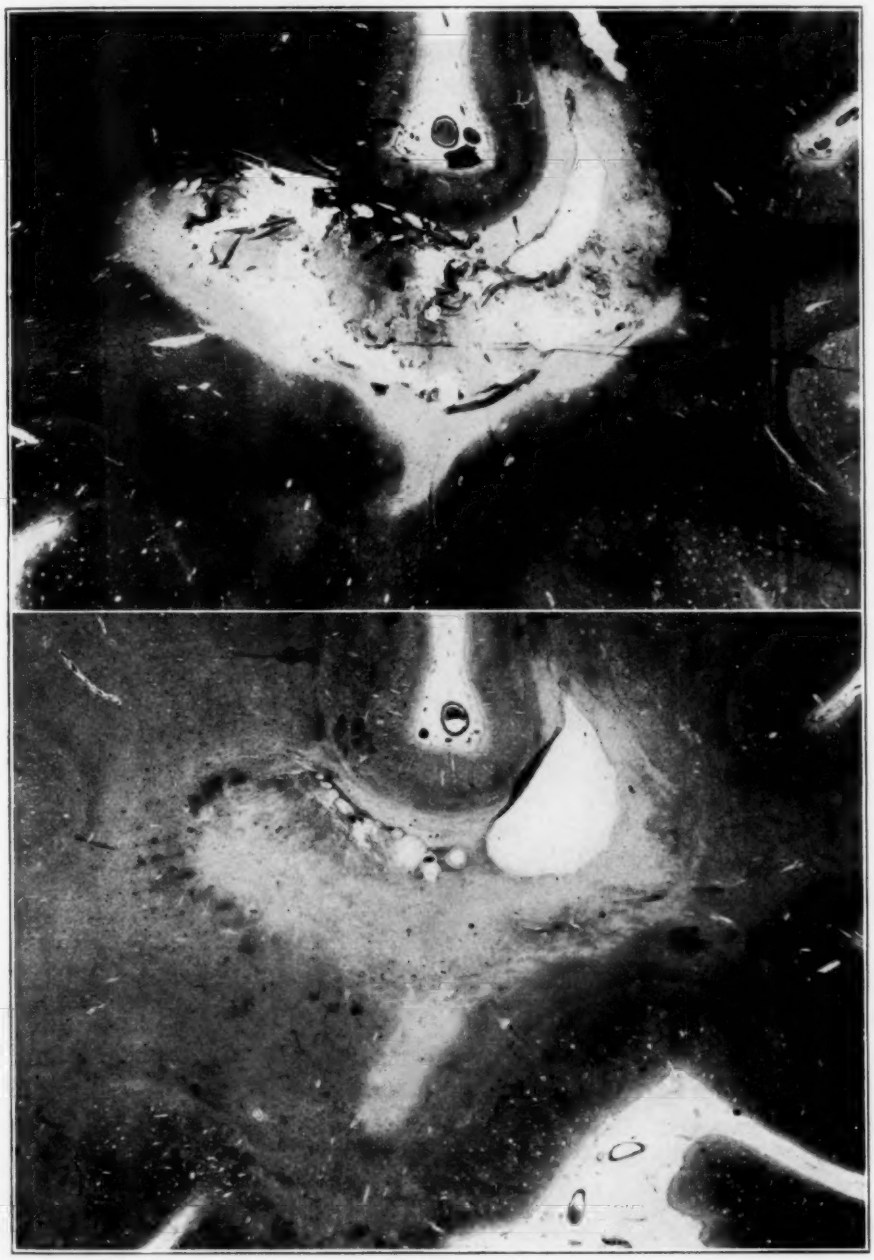

Fig. 2.—Sections from the same block taken from the tip of the occipital lobe in a case of multiple sclerosis. Above, Mahon stain for myelin sheaths; below, Bodian stain for axis-cylinders. Note extensive loss of axis-cylinders. Lens enlargement.

the brain stem (fig. 1), the loss of axis-cylinders was not recognizable at low magnifications because of the optical illusion, which has been discussed elsewhere.[9] With higher magnification some destruction could be observed. In other blocks, chiefly from the white matter of the hemispheres, the loss of axis-cylinders was striking even with the low and the medium power lens (figs. 2, 3 and 4), although there were some parts in most plaques in which loss of axis-cylinders was inconspicuous. In the same plaques, however, areas could be found in which high power fields showed a significant reduction in number of axis-cylinders—obviously a degree of loss of potential functional significance.

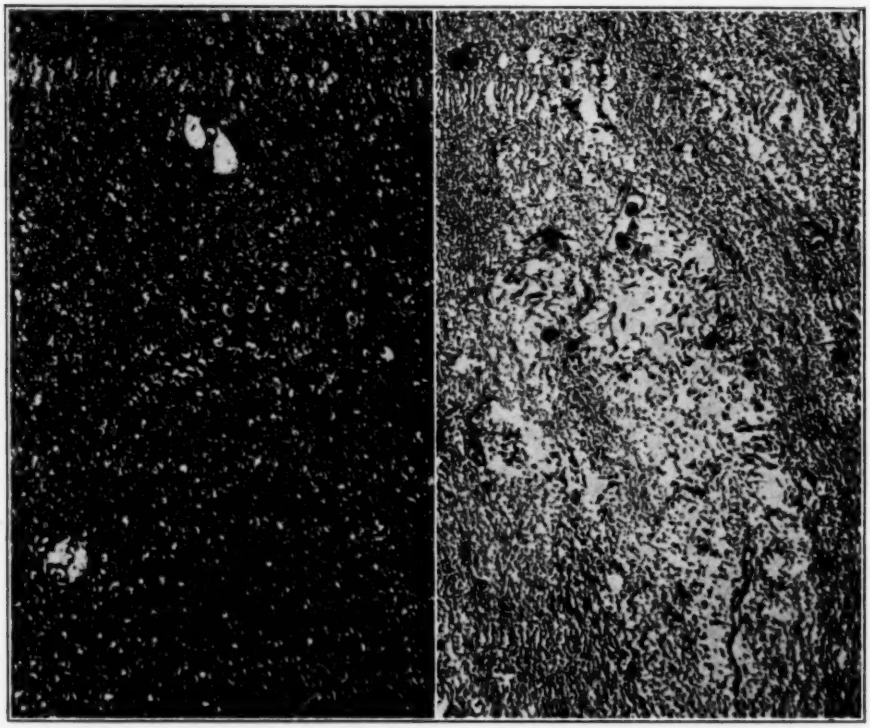

Fig. 3.—Axis-cylinders in relatively normal white matter (left) and in a periventricular plaque (right) from the same slide in an old case of multiple sclerosis. Bodian method; medium power lens.

An attempt was made to carry out the recommendation made in a previous paper,[9] that actual counts of axis-cylinders should be made. This proved difficult in many cerebral plaques because of the varying directions at which the fibers are running in the cerebral white matter and the resulting density of the feltwork in the relatively normal areas.

9. Alexander, L., and Putnam, T. J.: The Element of Optical Illusion in the Appearance of Preservation of Axis-Cylinders in Certain Lesions of the Central Nervous System, Arch. Neurol. & Psychiat. **44**:1312-1318 (Dec.) 1940.

This circumstance may cause the same fiber to be counted more than once in a given section. In the spinal cord, however, with its regular parallel arrangement of tract fibers, axis-cylinders and myelin sheaths can be counted in cross sections as easily as red blood cells in a blood-counting chamber.

An example of such a count is as follows: A section of spinal cord was chosen in which there was a definite sclerotic plaque extending exactly to the ventral septum, with relatively slight loss of axis-cylinders on casual inspection. This was stained by the Bodian method and photographed, together with the corresponding, relatively normal, side

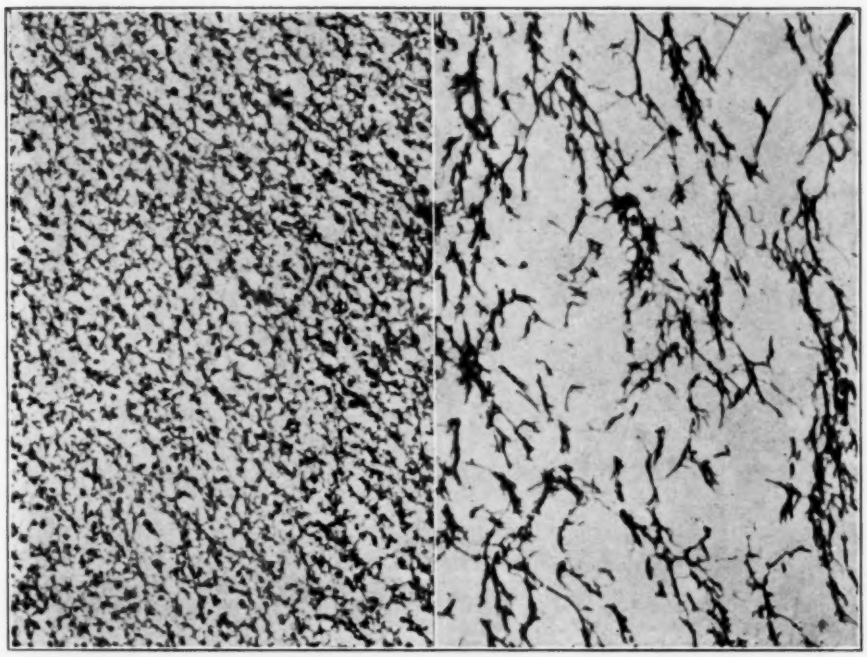

Fig. 4.—Axis-cylinders in relatively normal white matter (left) and in a periventricular plaque (right) from a case of multiple sclerosis. Bodian stain, Rosett modification; high power lens. The appearance under a low power lens suggested little loss of axis-cylinders.

(fig. 5 *A*). Also, two higher enlargements were taken of closely corresponding representative areas on each side of the septum (fig. 5 *B* and *C*). A segment of a micrometer objective was photographed at the same magnification, and from it squares measuring 0.01 mm. on each side were cut. These were used to enclose typical areas in each high power photomicrograph. Five such areas were counted on each side. The results were as follows: relatively normal side, 212, 216, 224, 235, 237 axis-cylinders per 0.01 mm. square, or 1,134 in all; area of plaque, 173, 176, 183, 189, 189 axis-cylinders per 0.01 mm. square, or 890 in all.

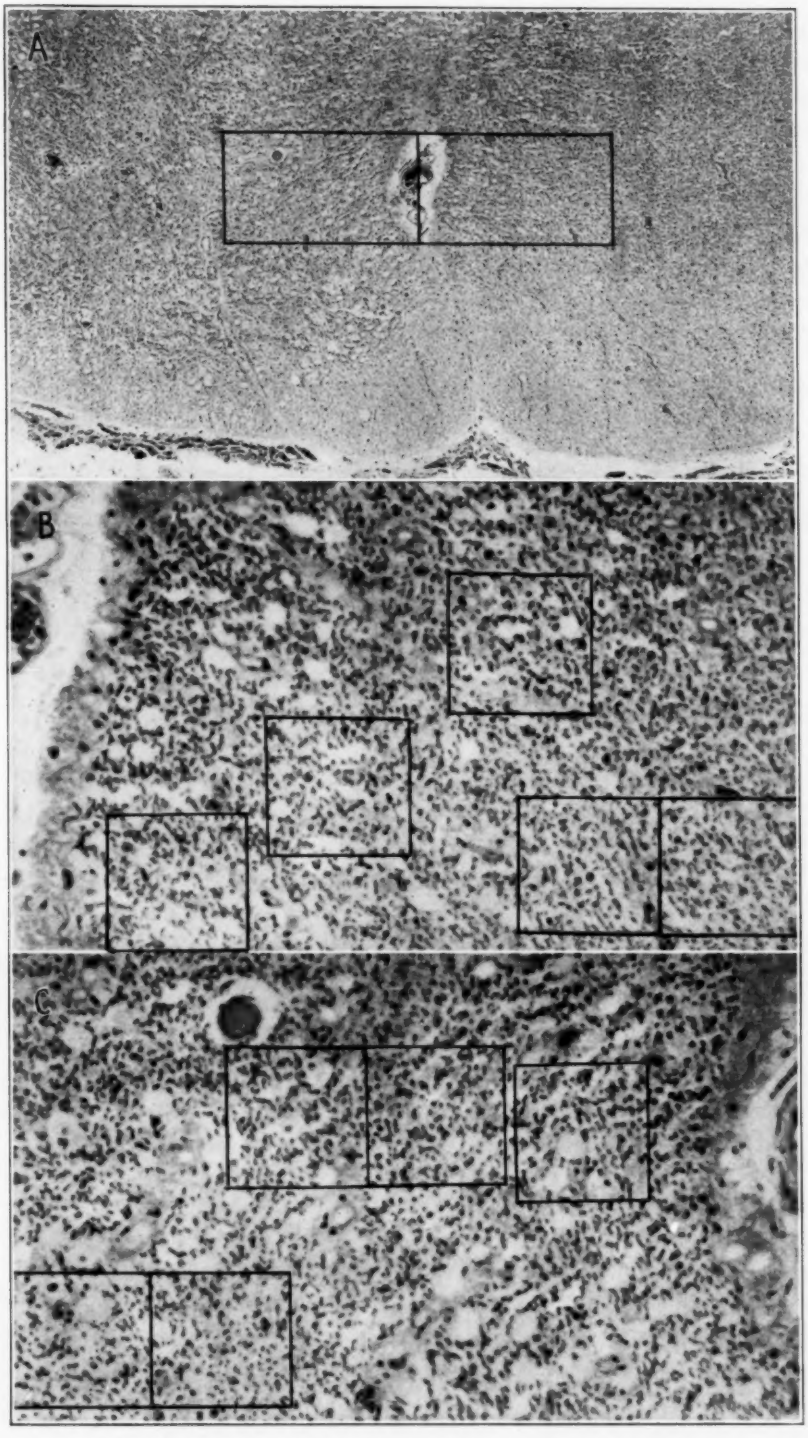


Fig. 5.—Section from the spinal cord in a case of multiple sclerosis, cut at 5 microns and stained by the Bodian method. *A*, low power view, showing at casual glance the apparent intactness of axis-cylinders on both sides and the areas enlarged in *B* and *C*. *B*, right side, relatively normal total number (1,134) of axis-cylinders in five 0.01 mm. squares; *C*; left side, area of plaque, total number (890) of axis-cylinders in five 0.01 mm. squares. The actual squares counted are drawn in (reduced).

In the more acute lesions, in which no fibrous gliosis had taken place, there was much interstitial edema and deformity of the axis-cylinders. Some of them were fragmented; others, thinned, tortuous or swollen. This was particularly striking in a case of the acute type sometimes described as neuromyelitis optica [10] (fig. 6). A group of abnormal, but persistent, axis-cylinders in this case, and the fact that axis-cylinders showing similar changes in experimental lesions apparently recover, suggest that this is a reversible change. These abnormalities were less conspicuous in the older lesions, but it was obvious that the average diameter of axis-cylinders was below normal. Usually the number of axis-cylinders actually missing was greater in the older than in the younger lesions.

On the whole, fibrous gliosis (as demonstrated with Mallory's phosphotungstic acid hematoxylin stain) was inconspicuous in all the cerebral plaques, and it was entirely absent in the acute ones.

*Secondary Degeneration.*—Another criterion of destruction of axis-cylinders is the production of secondary degeneration by intense lesions lying in regions where most of the fibers have a parallel course. Secondary degeneration of the pyramidal tracts in cases of multiple sclerosis has been reported by Dawson [11] and others. We have observed it occasionally. It did not occur in the case of neuromyelitis optica included in the series.

*Relation Between Loss of Myelin and Loss of Axis-Cylinders in Other Lesions of Vascular Origin.*—The previous quotation from Spielmeyer mentions the fact that a relative persistence of axis-cylinders may occur in any anoxic lesion of the nervous system. Few studies have been devoted to the subject, however. Farnell and Globus [12] called attention to the preservation of axis-cylinders in Binswanger's subcortical encephalopathy, obviously of vascular origin. A general review of the experimental [13] and pathologic aspects of the subjects [14] has been given elsewhere. In the course of one of these studies, lesions resulting from experimental blockage of cerebral veins by means of a bland oil were

10. Putnam, T. J., and Forster, F. M.: To be published.

11. Dawson, J. W.: The Histology of Disseminated Sclerosis, Tr. Roy. Soc. Edinburgh **1**:517-740, 1916.

12. Farnell, F. J., and Globus, J. H.: Chronic Progressive Vascular Subcortical Encephalopathy: Chronic Progressive Encephalitis of Binswanger, Arch. Neurol. & Psychiat. **27**:593-604 (March) 1932.

13. Putnam, T. J.: Studies in Multiple Sclerosis: Encephalitis and Sclerotic Plaques Produced by Venular Obstruction, Arch. Neurol. & Psychiat. **33**:929-946 (May) 1935.

14. Putnam, T. J., and Alexander, L.: Tissue Damage Resulting from Disease of Cerebral Blood Vessels, Proc. A. Research Nerv. & Ment. Dis., Proc. (1937) **18**:544-567, 1938.

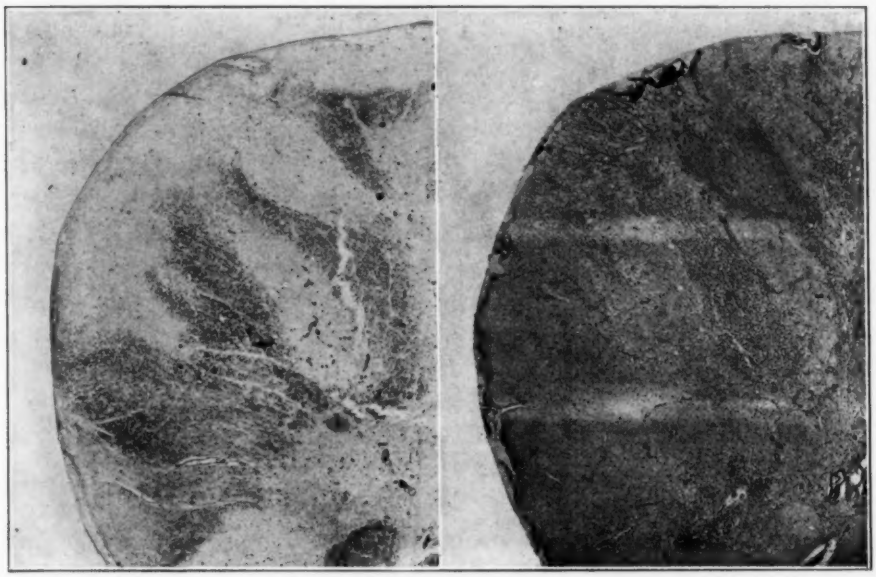

Fig. 6.—Sections from a block from the spinal cord in a case of multiple sclerosis of the neuromyelitis optica type. Left, Mahon stain for myelin; right, Bodian stain for axis-cylinders. Note apparent intactness of axis-cylinders. Lens enlargement.

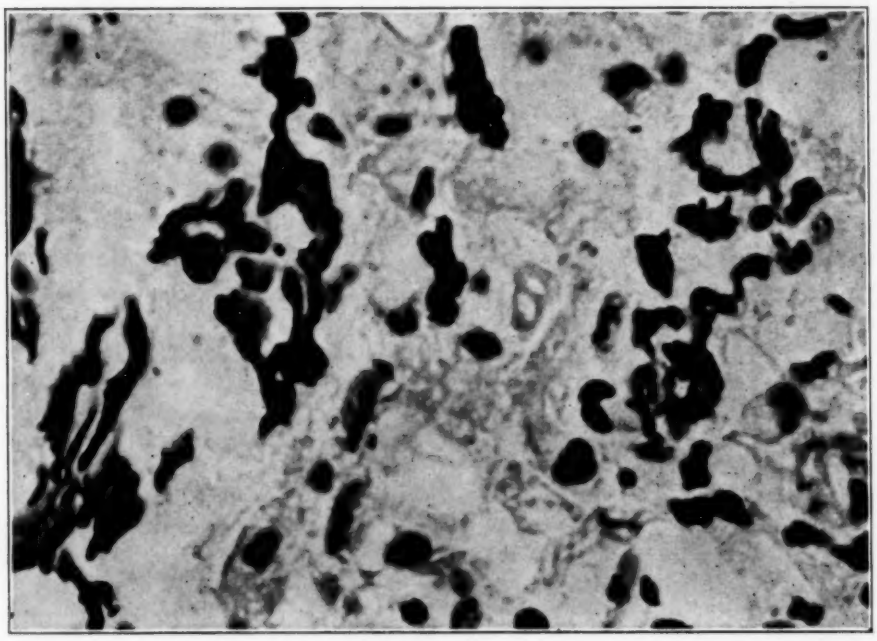

Fig. 7.—Swollen, hyalin-appearing axis-cylinders from the same slide as that shown in figure 6, right side. Bodian stain; oil immersion lens.

observed to consist of as exquisite a local demyelination with preservation of axis-cylinders as has even been encountered in pathologic material of human origin.[13]

In the course of the present study, 2 cases of cerebral arteriosclerosis, infarction and venous thrombosis, chosen at random, were studied in which small or large discrete lesions without actual softening had occurred. In all the lesions examined the degree of destruction of axis-cylinders showed the same range of variation as in the sclerotic plaques —that is, from the situation in which only a few axons per high power field are seen to that in which the density of axons appears altered only by swelling or by interstitial edema at low magnifications (fig. 7).

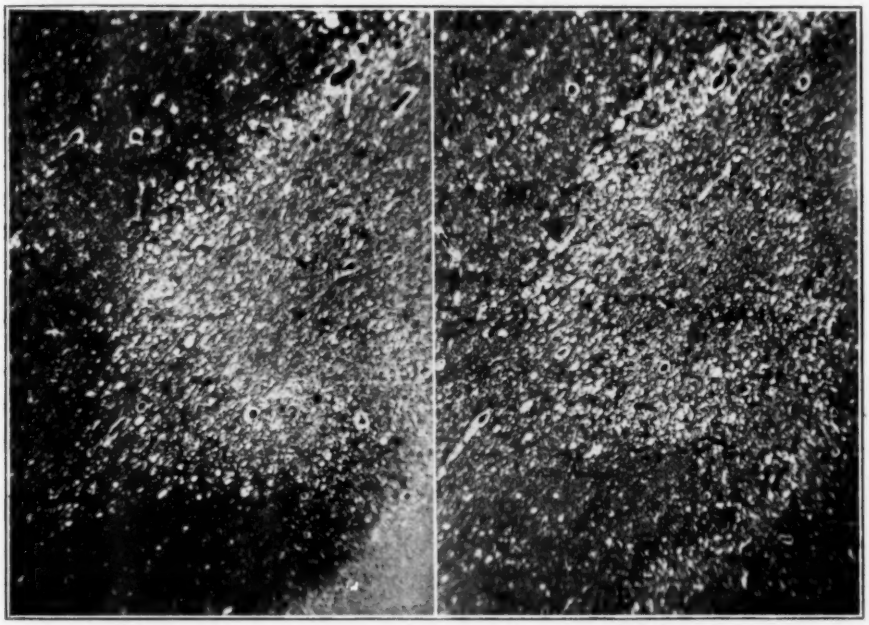

Fig. 8.—Sections from corresponding fields of adjacent sections from a case of cerebral arteriosclerosis. Left, Mahon stain; right, Bodian stain for axis-cylinders. Low power lens. Note the exquisite demyelination, with apparently relatively slight loss of axis-cylinders.

## COMMENT

The evidence here presented is obviously compatible with the point of view that has been presented elsewhere,[15] namely, that the parenchymal lesions in multiple sclerosis are secondary to a local disturbance of circulation. It suggests that the conception of a specific process of demyelination peculiar to multiple sclerosis deserves revision.

The fact that fibrous gliosis is not observed in the most acute lesions of multiple sclerosis strongly suggests that the glial scar is secondary

15. Putnam, T. J.: Evidences of Vascular Occlusion in Multiple Sclerosis and Encephalomyelitis, Arch. Neurol. & Psychiat. **37**:1298-1321 (June) 1937.

to the local destructive process. This is borne out by various earlier studies of this series [16] and by the more recent observations of Greenfield and King,[5] who never saw glial fibrosis in lesions less than two weeks old. Greenfield and King preferred a different interpretation of the process and objected to calling the gliosis secondary, but they apparently did not regard it as primary. The gliosis of sclerotic plaques is seldom as dense as that occurring in old secondary degenerations.[4]

The fact that many axis-cylinders are damaged in lesions of multiple sclerosis has also an important bearing on the problem of prognosis and treatment. It is reasonable to assume that the acute stage of swelling and distortion of axis-cylinders corresponds to the acute stage often observed in symptoms. There would appear to be little hope that any form of treatment would increase the number of persisting axis-cylinders once the lesion is formed. On the other hand, only an extension of the lesion would seem likely to injure substantially more axons than those seen damaged in the acute stage.

The clinical corollary is obvious: As has been pointed out elsewhere,[17] the effect of treatment should be estimated by its ability to prevent exacerbation in a significant proportion of cases over a sufficient period, rather than by the improvement in established symptoms which may coincide with its use.

## SUMMARY

Studies by means of the Bodian silver stain for axis-cylinders, which is uniquely specific and especially adapted to the purpose, showed that some degree of damage or destruction of axons occurred in all the sclerotic plaques investigated.

In all but 2 of the plaques examined the axis-cylinders were reduced to a small fraction of the normal number.

The pathologic appearance of surviving axis-cylinders was more striking in fresh plaques than in old ones.

Similar changes, of about the same degree of intensity, were seen in some other lesions of vascular origin, not sufficiently intense to cause complete necrosis.

The clinical correlation of these observations, especially with respect to treatment, is discussed.

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16. Putnam (footnotes 4, 13 and 15).

17. Putnam, T. J.: The Criteria of Effective Treatment in Multiple Sclerosis, J. A. M. A. **112**:2488-2491 (June 17) 1939.

# TOPOGRAPHIC DISTRIBUTION OF LESIONS IN CENTRAL NERVOUS SYSTEM IN JAPANESE B ENCEPHALITIS

## Nature of the Lesions, with Report of a Case on Okinawa

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CINCINNATI

THE OBJECT of this study was to determine the nature and distribution of various types of lesions in the central nervous system of a patient with Japanese B encephalitis. The case was one that occurred during an outbreak of this disease on Okinawa in the summer of 1945.

## REPORT OF CASE

### CLINICAL HISTORY

A young white American soldier was admitted to a field hospital on Okinawa on Aug. 4, 1945, in a somnolent state. From his fragmentary story, it appeared that he had become ill about July 28, with headache, fever, pain in the eyes, weakness and dizziness. He stated that he felt "mixed up," that the headache was "terrible" and that during the previous week he had become increasingly drowsy.

On admission, seven to eight days after the onset of illness, his temperature was 104 F. and his pulse rate 100 a minute. He was cooperative one moment and irritable the next. He kept clutching at the bedclothes. He could be awakened with difficulty for questioning but soon lapsed again into a deep sleep. He was unable to state the name of his unit, where he was, the day of the week or the date. His neck was rigid, and pain was elicited on forward flexion of the head. There was no evidence of involvement of the cranial nerves. The optic disks appeared normal. The biceps and triceps reflexes were diminished on both sides; the knee jerks were equal and hyperactive, and the ankle jerks were normal. The abdominal reflexes were hyperactive; the cremasteric reflexes were diminished. The Babinski sign could not be elicited. The cerebrospinal fluid contained 192 cells per cubic millimeter, of which 97 per cent were of mononuclear type, and

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From the Army Institute of Pathology, Washington, D. C., and the Children's Hospital Research Foundation, Department of Pediatrics, University of Cincinnati College of Medicine.

* The work on Okinawa, and subsequently in the United States, was done while Dr. Sabin was serving as lieutenant colonel in the Medical Corps, Army of the United States, with the Army Epidemiological Board, Preventive Medicine Service, Office of the Surgeon General, United States Army. The investigation was sponsored by the Commission on Neurotropic Virus Diseases, Army Epidemiological Board.

the Pandy test was reported to give a negative reaction. The leukocyte count of the blood was 10,650, with 77 per cent neutrophils and 23 per cent lymphocytes. No malarial parasites were seen in the blood smear.

On August 5 the patient was completely disoriented, lethargic and rigid. The axillary temperature was 101.6 F., and the pulse rate was 88. Two generalized convulsions occurred. The abdominal reflexes were now absent, and the knee and ankle jerks were exceedingly hyperactive. Ankle clonus was readily elicited. The Hoffmann sign was not elicited. Further examination was not made because of the patient's rigidity and extreme restlessness. He died at 3 a. m. on August 7, about ten days after onset of the illness. Major Wiley L. Forman, of the Medical Corps, supplied the clinical observations on this patient.

*Necropsy.*—Necropsy was performed five hours after death. Examination of the viscera, by Capt. Robert A. Green and Lieut. Benjamin F. Levy, of the Medical Corps, revealed nothing noteworthy except for slight enlargement of the spleen. The brain and the spinal cord were removed by one of us (A. B. S.) under relatively aseptic conditions. Grossly, the external surface of the brain showed no change other than diffuse congestion. The spinal cord was generally rather soft, and in the lower cervical region was almost mushy.

## VIRUS STUDIES

Under sterile conditions, blocks of gray matter, approximately 1 cm. in size, were removed from the frontal, parietal and occipital cortex of both sides, and smaller pieces were taken from the midbrain, pons, medulla oblongata, cerebellar cortex and upper cervical portion of the cord. The tissue was placed in 50 per cent buffered glycerin in saline solution, stored immediately in a refrigerator, then packed in ice and transported to a field laboratory, where at about 5 p. m. of the same day an extract was inoculated into mice.

Although attempts to isolate the virus were unsuccessful, despite the large numbers of lesions observed histologically, it seems desirable to record in detail the procedure employed. One half of each piece of tissue removed was used and the rest saved. What was used was washed only once in saline solution, and about 4 Gm. was ground without abrasive, in 10 cc. of saline solution. The concentrated extract obtained after a short period of centrifugation was injected intracerebrally (0.03 cc.) and intra-abdominally (1.0 cc.) into 5 Swiss albino mice brought from the United States. These mice were kept in screened cans and observed daily. On the fifteenth day after inoculation, 1 of the mice which exhibted a reddish discharge around both eyes, had ruffled fur and in other ways appeared ill, was killed. Part of its brain was passed immediately through 5 more mice, and the rest was stored in 50 per cent buffered glycerin in saline solution in the cold and subsequently was taken to the United States, where on Sept. 21, 1945, it was passed through another 5 mice. The remainder of the original human tissue, after storage in the refrigerator for eleven days, was thoroughly washed in saline solution, and a suspension of it was inoculated into another group of 5 Swiss mice, brought from the United States. All these attempts at inoculation yielded negative results. The subsequent disclosure of widespread cerebral lesions in this case warrants the assumption that the samples taken for animal inoculation were similarly affected but that the lesions no longer contained virus. It has previously been demonstrated [1] in cases of human polio-

1. Sabin, A. B., and Ward, R.: Nature of Non-Paralytic and Transitory Paralytic Poliomyelitis in Rhesus Monkeys Inoculated with Human Virus, J. Exper. Med. **73**:757-770, 1941.

myelitis that when death occurred three to four days after onset of paralysis the virus could not be isolated from the spinal cord, medulla oblongata, midbrain or diencephalon, where the lesions were advanced, but could still be obtained from the motor cortex and the contents of the descending colon.

An attempt to isolate virus from cerebrospinal fluid obtained from the patient about eight days after the onset, or two and a half days before death, also yielded negative results. Tests on blood serum taken at the same time indicated that, while complement-fixing antibodies were absent, small but definite amounts of neutralizing antibodies for the Okinawa strain of Japanese B encephalitis virus (isolated from another fatal case [2]) were present. The results shown in the table indicate that while the neutralization index in the intracerebral test was only 32, that in the intraperitoneal test was at least 10,000. One of us (A. B. S.) has

*Neutralization Tests with Serum of the Patient* and Japanese B Encephalitis Virus (Okinawa Strain)*

| Type of Test | Source of Serum | Dilution of Virus in Mixture | | | | | | | $LD_{50}$ Titer of Virus | Neutralization Index † |
|---|---|---|---|---|---|---|---|---|---|---|
| | | $10^{-3}$ | $10^{-4}$ | $10^{-5}$ | $10^{-6}$ | $10^{-7}$ | $10^{-8}$ | $10^{-9}$ | | |
| Intracerebral | Normal rabbit control..... | ... | ... | ... | 4/4‡ | 3/4 | 3/4 | 0/4 | $10^{-8.2}$ | ..... |
| | Patient..... | ... | ... | ... | 3/4 | 1/4 | 1/4 | ... | $10^{-6.7}$ | 32 |
| Intraperitoneal (14 day old mice) | Normal rabbit control..... | 4/4 | 4/4 | 3/4 | 3/3 | 3/4 | 3/4 | ... | $10^{-8.2}$ +? | ..... |
| | Patient..... | 2/3 | 2/4 | 1/4 | 1/4 | 0/3 | ... | ... | $10^{-4.2}$ | 10,000 (+?) |

* The serum was obtained seven to eight days after onset of the illness, and two and a half days before death.

† Neutralization index $= \frac{LD_{50} \text{ titer of virus in control serum mixtures}}{LD_{50} \text{ titer of virus in test serum mixtures}}$.

‡ 4/4 indicates that all 4 inoculated mice died of encephalitis; 3/4 that 3 mice died, etc. Similarly, 3/3 indicates that all 3 inoculated mice died, etc.

found that when a very low, questionable neutralization index in the intracerebral test is due to nonspecific factors the intraperitoneal test yields negative results. However, a human serum obtained about three days after onset of Japanese B encephalitis has been found to have an intracerebral neutralization index of 20, whereas in the intraperitoneal test the index was at least 500,000.

## MATERIAL AND METHODS FOR HISTOLOGIC STUDY

The material consisted of the entire brain and spinal cord, portions of the peripheral nervous system, the pituitary gland and representative blocks from the thoracic and abdominal viscera. All had been fixed in solution of formaldehyde U. S. P. (1:4).

Since the study was undertaken to gain an understanding of the topographic distribution of the lesions of Japanese B encephalitis, representative blocks of tissue were removed from every level of the central nervous system bilaterally. The

2. Sabin, A. B.: Outbreak of Encephalitis on Okinawa in 1945: Preliminary Report on Status as of 27 August 1945, J. Mil. Med. in Pacific **1**:79-84, 1945; Epidemic Encephalitis in Military Personnel: Isolation of Japanese B Virus on Okinawa in 1945, Serologic Diagnosis, Clinical Manifestations, Epidemiologic Aspects and Use of Mouse Brain Vaccine, J. A. M. A. **133**:281-293 (Feb. 1) 1947.

3. Footnote deleted by authors.

cerebral cortex was generously sampled, one or two blocks being taken from each gyrus. From the caudate and lenticular nuclei and the diencephalon the sampling was done at four levels; from the hippocampal formation, at three levels, and from the nucleus amygdalae, at two levels. Two blocks each were obtained from the

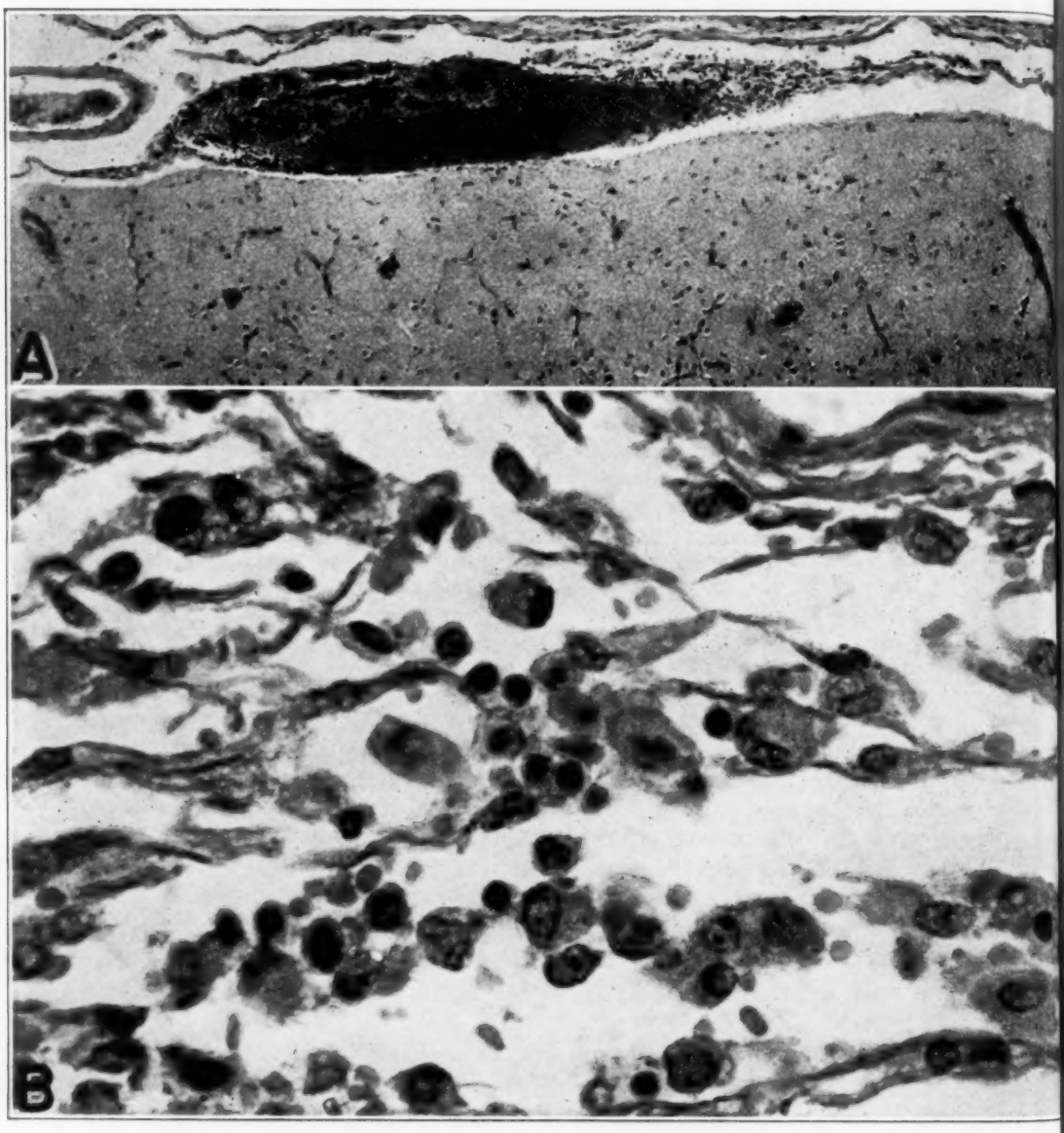


Fig. 1.—Cellular constituents of the leptomeninges. *A* (× 100; AIP neg. 92622), from the medial orbital gyrus, shows a vein surrounded with lymphocytes, some of which trail off into the adjacent arachnoidal meshes. A few larger cells in the arachnoid, having the characteristics of histiocytes, also are visible. In *B* (× 860; AIP neg. 93063), from the leptomeninges of the precentral gyrus, some of the small cells may be lymphocytes, but the majority probably represent stages in the development of trabecular cells into histiocytes, of which many are present in the arachnoid meshes. Hematoxylin and eosin stain.

midbrain, the pons and the medulla oblongata, and six, from the spinal cord. Altogether, nine blocks were taken from the cerebellum, three of which contained portions of the dentate or the roof nuclei. The choroid plexuses were included in samples removed from the region of the ventricles.

All the blocks of tissue used in the topographic study were embedded in paraffin, sectioned at 6 microns and stained with hematoxylin and eosin. Additional blocks were embedded in pyroxylin and stained with cresyl violet. Frozen sections were

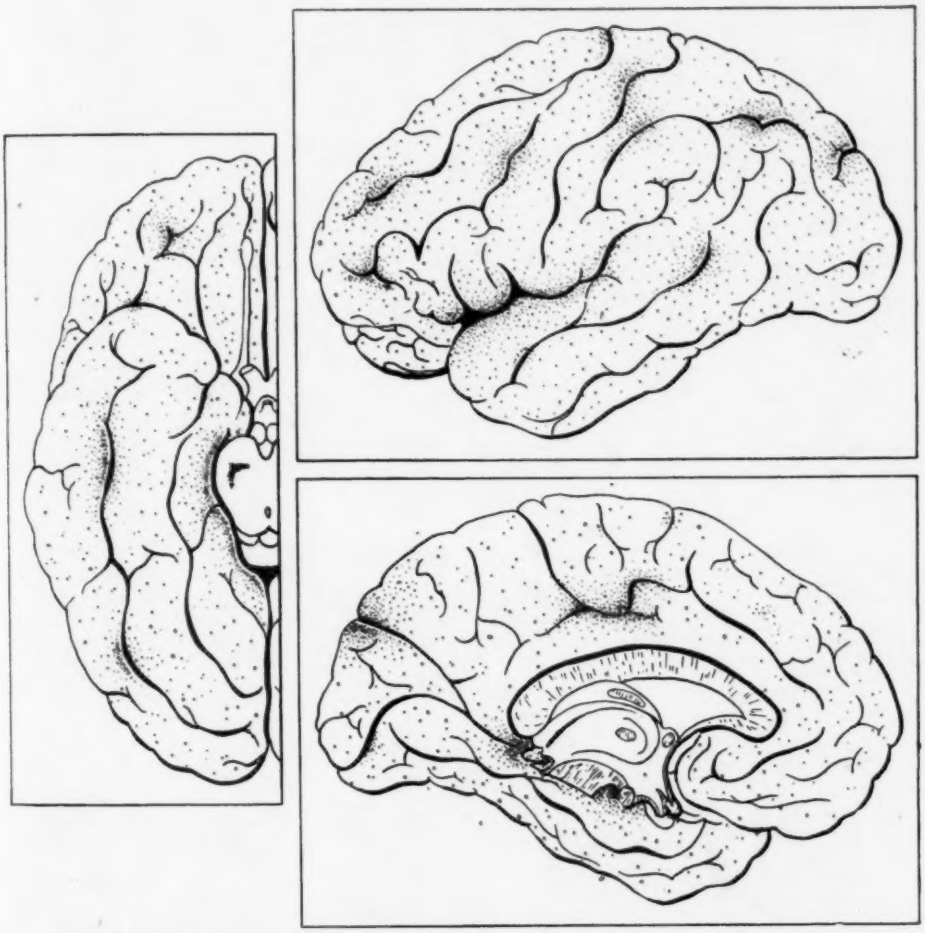

Fig. 2.—Distribution of leptomeningeal cells, both exudative and reactive, over the three surfaces of the brain.

impregnated with silver by the Hortega method and were stained for fat by means of sudan III and for myelin by a modification of the Weigert method.

Preliminary examination of the sections of the brain revealed diffuse involvement of the leptomeninges and a multitude of nodules in the gray matter. In an effort to determine the relative degree of involvement at the various levels of the central nervous system, the following procedure was adopted: Beginning with the cerebral cortex, the number of low power fields (magnification, 35) in

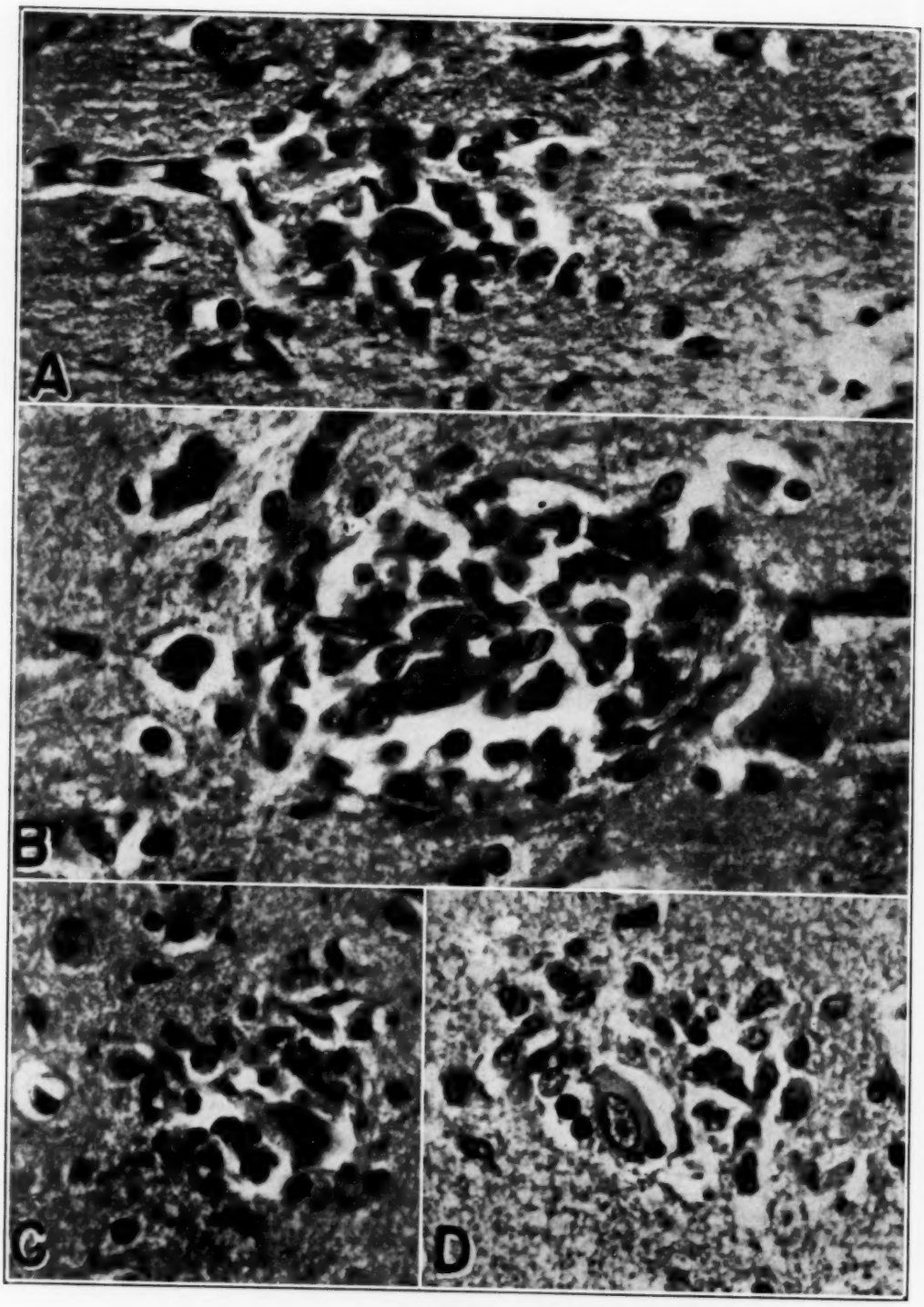


**Figure 3**

*(See legend on opposite page)*

each section was determined; then the nodules in all the fields were counted, and from these two figures the number of nodules in each low power field of the cerebral cortex was ascertained. The counts were made first on one side of the brain and then on the other, and an average was struck. The same calculations were carried out in sections of the subcortical white matter. When lesions of the cerebral cortex were diffuse, occupying a lamina or two, they were regarded as the equivalent of two to four nodules. Then the leptomeninges were examined, and the degree of cellular increase was estimated in terms of 1 to 4 plus. The same evaluation was made with respect to inflammatory cells surrounding blood vessels of the cerebral cortex and the subcortical white matter.

The degree of involvement of the cerebral cortex served as the basis for estimating the density of the lesions in the rest of the brain and in the spinal cord. So great were the difficulties entailed in counting the number of microscopic fields in the various areas of gray matter that it was necessary to rely on a visual impression of the number of nodules and of perivascular cuffs of lymphocytes in the respective structures.

## HISTOLOGIC OBSERVATIONS

*Cellular Constituents in the Leptomeninges and Their Distribution.*—The cells occupying the leptomeninges were predominantly lymphocytes and histiocytes. The lymphocytes, with a few plasma cells intermixed, were mainly perivenous in location (fig. 1 *A*), but some were scattered through the meshes of the arachnoid. Perivenous cuffs of lymphocytes frequently extended downward in the Robin-Virchow spaces of cortical veins. The aggregations of lymphocytes were somewhat more numerous within sulci and fissures than over the surface of the brain. The histiocytes, on the other hand, were usually independent of vessels, collections of them lying free in the meshes of the arachnoid. Appearances suggested that many arose in the arachnoid, since all gradations, from hyperplastic trabecular cells to well developed histiocytes, were present (fig. 1 *B*). Phagocytosis of lymphocytes and unidentified debris by histiocytes was frequently observed. Accumulations of histiocytes were most common at the mouth and the base of fissures and sulci.

The over-all distribution of the leptomeningeal reaction is shown in figure 2. The spread was uniform on the three surfaces of the brain, except for concentration in the region of a number of the sulci and fissures. Leptomeningeal reaction over the hypothalamic region and in the interpeduncular fossa was of moderate degree, but was minimal over the midbrain, pons, medulla oblongata, cerebellum and spinal cord. None of the leptomeningeal blood vessels were thrombosed or showed intimal proliferation.

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Fig. 3.—Neuronophagic nodules in various stages of development. In *A* (× 625; AIP neg. 92618), from the inferior frontal gyrus, a necrotic ganglion cell is surrounded with pleomorphic mononuclear cells, some of which are emerging from the perineuronal space; similar cells line the edges of the adjacent vessel. In *B* (× 700; AIP neg. 92616), from the same gyrus, appearances suggest that the perineuronal cells may, in part, have arisen from the vessel skirting the left edge of the neuronophagic nodule. In *C* (× 600; AIP neg. 92615), from the precuneus, the perineuronal cells are beginning to invade the parenchyma; the nuclei of most of the cells are irregularly elongated. In *D* (× 675; AIP neg. 94492), from the superior frontal gyrus, it appears likely that the reactive cells lying in the parenchyma adjacent to an intact ganglion cell may have arisen at a level somewhat removed from that photographed. Three oligodendrocytes are in the perineuronal space. Hematoxylin and eosin stain.

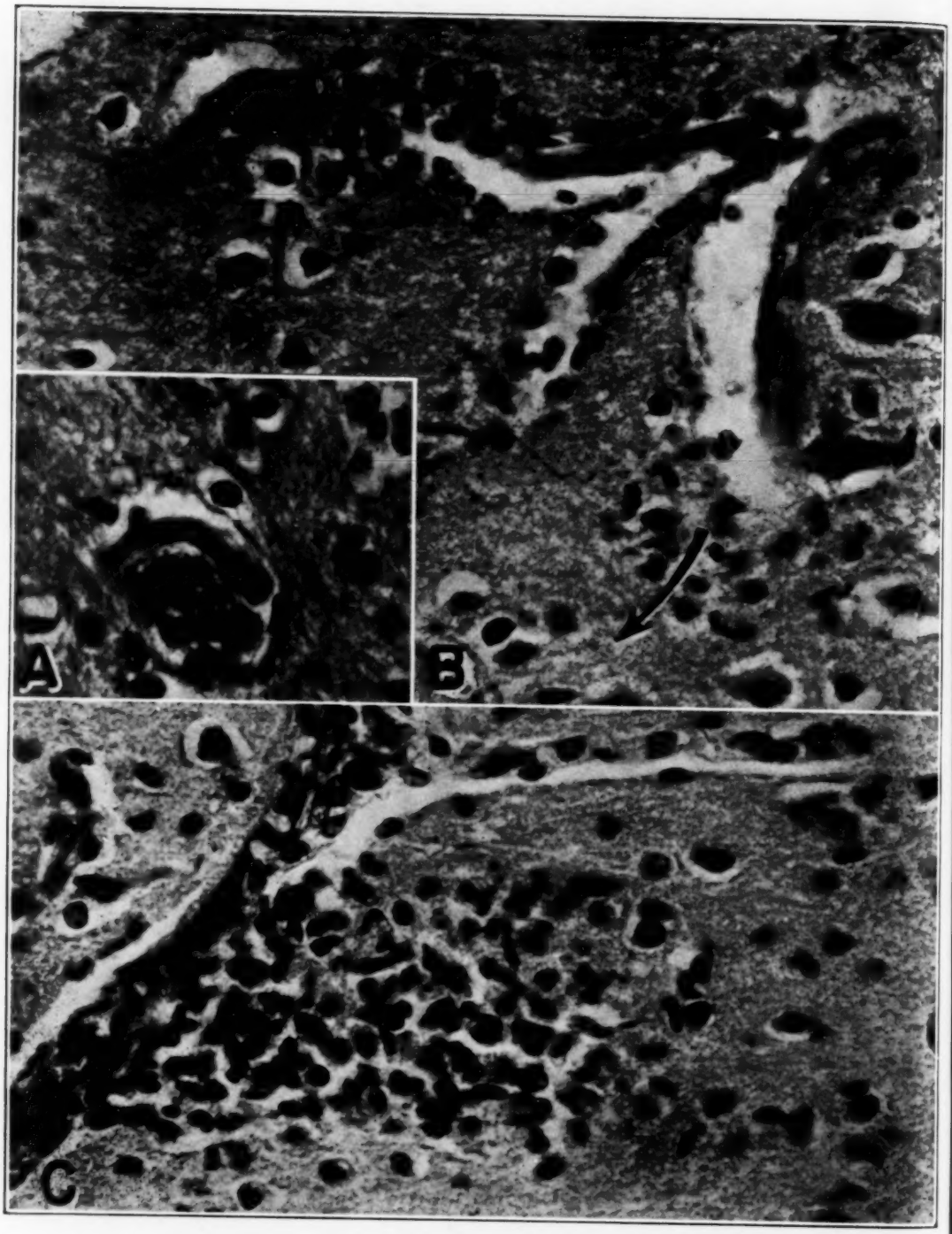


Figure 4

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Samples of the dura revealed no abnormalities except for an occasional hemorrhage and scattering of lymphocytes in some of the arachnoidal granulations.

*Types of Lesions in the Central Nervous System and Their Distribution.*—Two types of lesions predominated in the central nervous system: nodules and perivenous cuffs of lymphocytes. These lesions were restricted to the gray matter except for the perivenous cuffs of lymphocytes, which were present to a limited degree in the subcortical white matter.

Nodules of diverse size permeated the gray matter. In the cerebral cortex they were in close relation to degenerated or necrotic ganglion cells and to the walls of blood vessels. Many arising in the vicinity of ganglion cells (neuronophagic nodules) were confined to perineuronal spaces (fig. 3 *A*), sometimes ballooning the space to an extraordinary size (fig. 3 *B*) and frequently extending into the surrounding parenchyma (fig. 3 *C*). Occasionally there were nodules in the parenchyma which surrounded relatively normal ganglion cells (fig. 3 *D*); it is possible that these arose at a level somewhat removed from the one illustrated. The nodules related to blood vessels also varied in size, the smallest consisting of a few cells within the pericapillary spaces (fig. 4 *A*). Appearances indicated that the cells invaded the parenchyma at focal points (fig. 4 *B*), continuing to develop until relatively large nodules were formed (fig. 4 *C*). Often there was no clue as to the origin of larger nodules (fig. 5 *A*).

The cellular composition of all the nodules, whether arising in proximity to ganglion cells or to blood vessels, was essentially the same. Lymphocytes, characterized by a densely chromatic, spherical nucleus and scanty cytoplasm, were generally rather scant and tended to be most numerous in perivascular spaces (fig. 4 *B*). Occasionally cells resembling lymphocytes appeared to be in the process of passing through a vessel wall. However, the predominant cell was of the large mononuclear type, with a moderately chromatic, spherical, ovoid or irregularly elongated nucleus and rather abundant cytoplasm. Whether the majority issued from the blood stream or arose from fixed tissue cells of the adventitia could not be decided from microscopic examination. Appearances suggested that some of the cells within perineuronal spaces may have originated from an adjoining vessel (fig. 3 *B*), but it is plausible to assume that some arose from fixed tissue cells lining the perineuronal space. Hortega microglia cells were absent from the nodules, but cells advancing into the parenchyma (figs. 3 *C* and 4 *B*), and even those within perineuronal spaces (fig. 3 *B*), had pleomorphic nuclei, some of which bore a resemblance to the nuclei of Hortega cells. No gitter cells were observed, and sections of cerebral cortex stained with sudan III disclosed fat globules in only a few perivascular cells and in moderate numbers of ganglion cells. A search for neutrophils in the cerebral cortex was fruitless. Nowhere did astrocytes participate significantly in the reaction. Oligodendroglial satellitosis about ganglion cells was commonly observed in the deeper laminas of the cerebral cortex (fig. 5 *C*).

Fig. 4.—Stages in development of perivascular nodules. In *A* (× 700; AIP neg. 92620), from the precentral gyrus, there is a clump of pericapillary cells which presumably are histiocytes arising in situ. In *B* (× 550; AIP neg. 92617), from the inferior frontal gyrus, the perivascular cells, comprising mononuclear cells, are in the process of invading the parenchyma (see arrow). In *C* (× 500; AIP neg. 92625), from an orbital gyrus, the parenchyma is being invaded by a mass of cells, mainly of the large mononuclear type. Contiguous perivascular spaces are heavily populated with cells, which are presumed to be both hematogenous and adventitial. Hematoxylin and eosin stain.

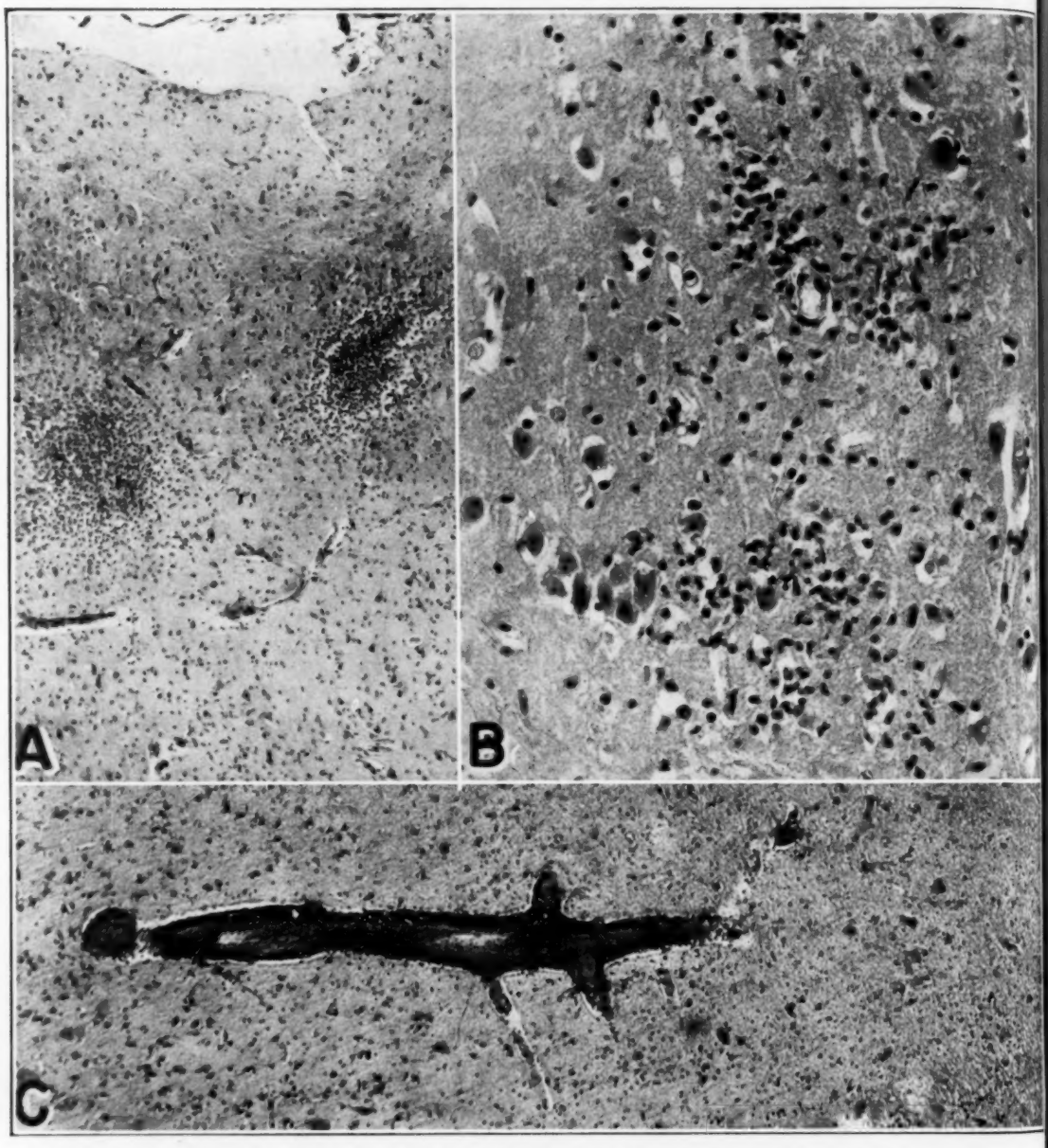



Fig. 5.—In *A* (× 69; AIP neg. 92624), from the anterior part of the cingular gyrus, there are two large nodules, composed chiefly of mononuclear cells. A search failed to reveal neutrophils. In *B* (× 210; AIP neg. 94457), from the hippocampal gyrus, diffuse aggregations of polymorphic mononuclear cells are concentrated around deteriorating ganglion cells and in regions from which ganglion cells have disappeared. In *C* (× 64; AIP neg. 92609), from the superior temporal gyrus, a vein at the corticomedullary junction is surrounded by a collar of lymphocytes. Oligodendroglial satellitosis is prominent. Hematoxylin and eosin stain.

In addition to the relatively discrete nodules, there were areas, involving a lamina or two and including numerous neuronophagic nodules, which were permeated with cells similar to those described in the preceding paragraph (fig. 5 *B*).

The cerebral white matter was but little affected. Perivenous cuffs of lymphocytes predominated in the zone of the corticomedullary junction (fig. 5 *C*). Occasionally there were proliferated oligodendrocytes and mononuclear cells, mainly perivascular in distribution. Neither stains for myelin nor those for fat revealed abnormalities.

The distribution of nodules over the three surfaces of the cerebral cortex is illustrated in figure 6. The numbers give the estimated number of nodules per

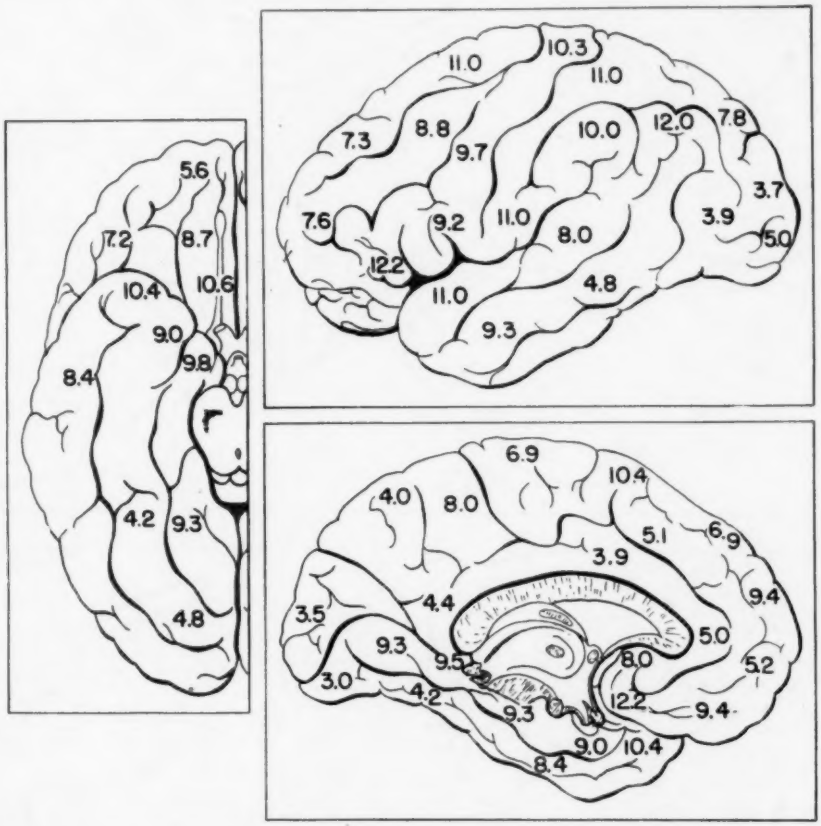



Fig. 6—The three surfaces of the brain, showing the number and distribution of nodules in the cortex. The figures indicate the number of nodules per field at magnification of 35 diameters.

low power field (magnification, 35). They tended to be most numerous in the region of the lateral fissue, the upper convex surface of the brain, the tip of the temporal lobe and the hippocampal and lingual gyri. The occipital lobe contained the fewest. The nodules were present in all laminas of the cerebral cortex, being least numerous in the uppermost.

The relative density of nodules and perivenous accumulations of lymphocytes in transverse sections of the brain, brain stem, spinal cord and cerebellum is illustrated in figure 7. The cuffs of lymphocytes predominated at the cortico-

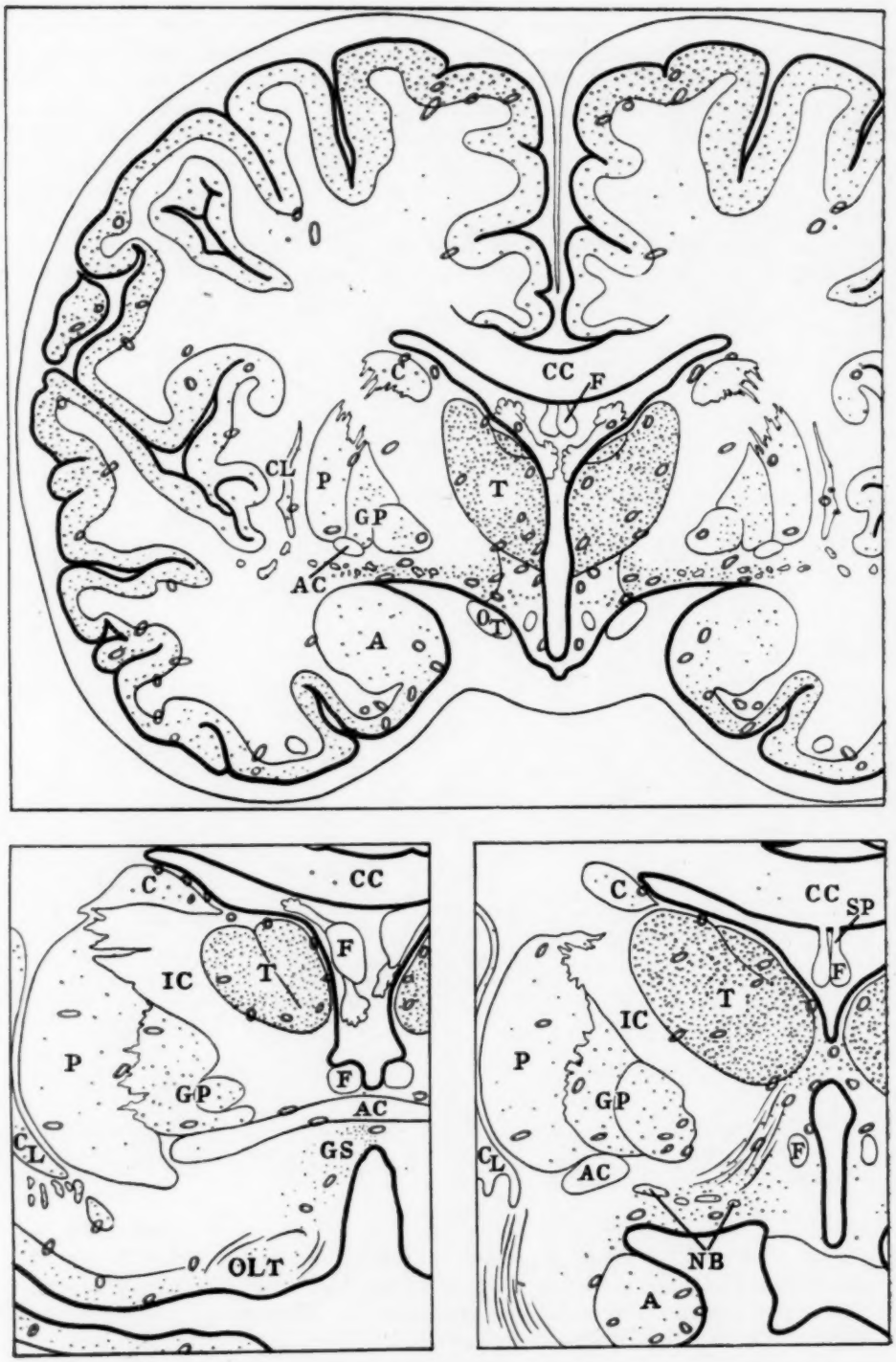
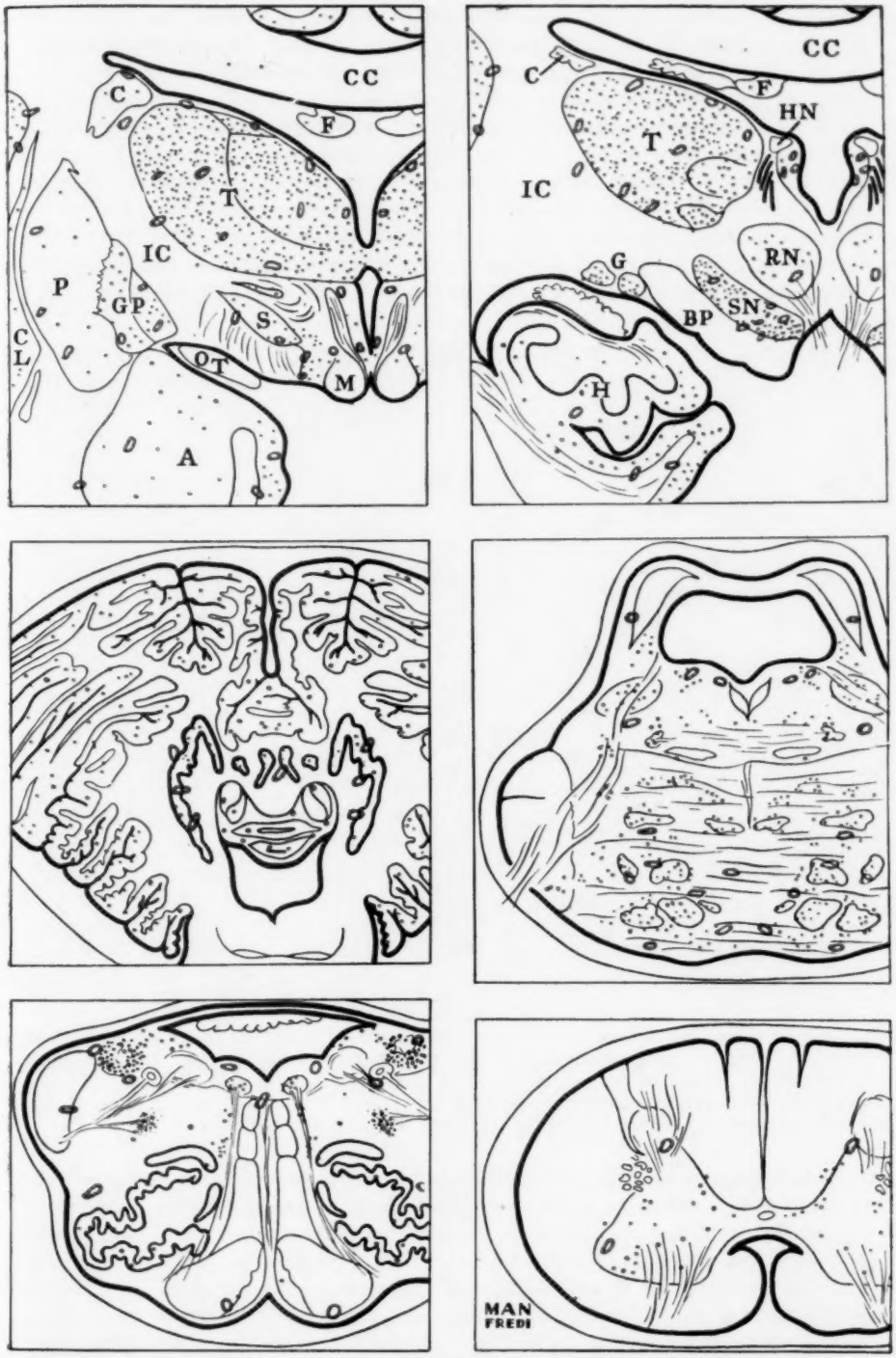



Fig. 7.—Distribution of nodules and perivenous cuffs of lymphocytes in the brain and spinal cord. In some regions, notably the thalamus and the substantia nigra, the nodules tended to be confluent. *A* is nucleus amygdalae; *AC*, anterior commissure; *BP*, basis pedunculi; *C*, caudate nucleus; *CC*, corpus callosum;




*CL*, claustrum; *F*, fornix; *G*, geniculate bodies; *GP*, globus pallidus; *GS*, gyrus subcallous; *H*, hippocampus; *HN*, habenular nuclei; *IC*, internal capsule; *M*, mamillary body; *NB*, nucleus basalis; *OLT*, olfactory tubercle; *OT*, optic tract; *P*, putamen; *RN*, red nucleus; *S*, subthalamic body; *SN*, substantia nigra; *SP*, septum pellucidum; *T*, thalamus.

medullary junction and in the periventricular region adjacent to the head of the caudate nucleus, the thalamus and the habenula. The relative number of nodules in the cerebral cortex shown in figure 7 is based on the numerical estimates indicated in figure 6. The diagrams of the transverse sections illustrate that, so far as the basal ganglia were concerned, the thalamus bore the brunt of the attack. It was riddled with nodules and permeated diffusely with cells, and its vessels were frequently surrounded by lymphocytes. Microscopic examination revealed that many, if not the majority, of the nodules were neuronophagic, most of the cells within pericellular spaces being mononuclear. Invasive cells in the parenchyma were of similar appearance; in addition, there were clumps of neutrophils, which sometimes were perivascular in location. Numerous ganglion cells had disappeared.

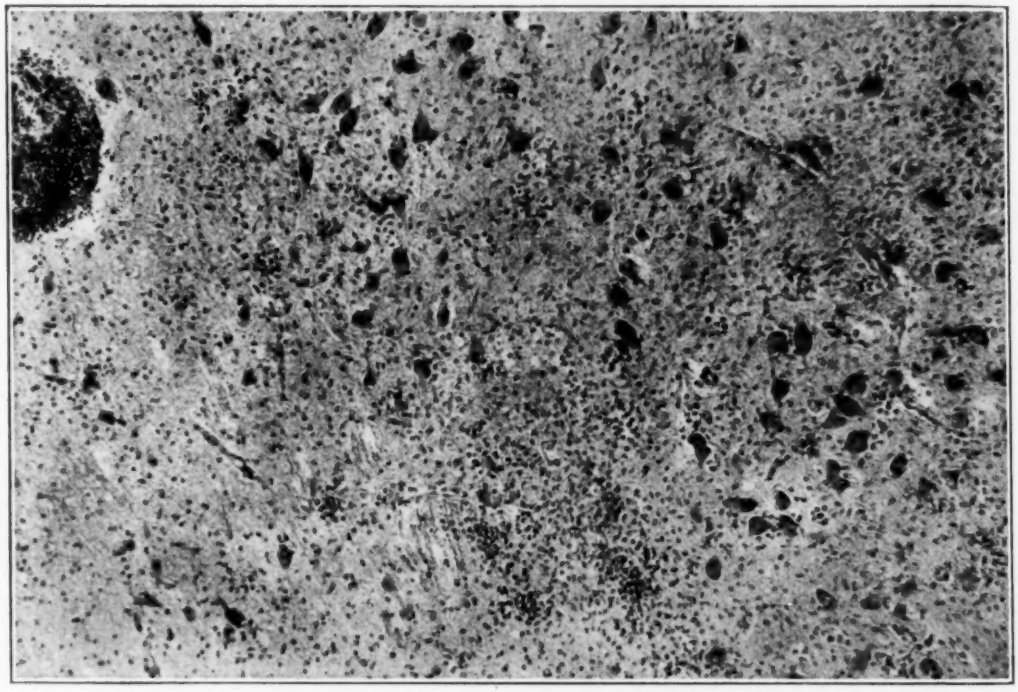


Fig. 8.—Substantia nigra, showing confluence of nodules, many of which are neuronophagic. Numerous ganglion cells are in a disintegrated state or have disappeared. Small clumps of neutrophils were observed in this section. A large perivenous collection of lymphocytes is present in the upper left corner of the photomicrograph. Hematoxylin and eosin stain (× 90; AIP neg. 92685).

The nucleus basalis and the globus pallidus were among the more extensively involved cerebral structures, and after them were the lateral hypothalamic nuclei, the geniculate bodies, the subthalamic nuclei, the hippocampus, the nucleus amygdalae and the olfactory tubercle, in that order. Only in the nucleus amygdalae was there a relatively large area of focal necrosis, at the periphery of which a proliferation of microglia cells had taken place. In the hippocampus, the nodules were strewn in the pyramidal layer (fig. 5 *B*) and left virtually untouched the gyrus dentatus. Diffuse aggregations of cells sometimes included oligodendrocytes and microglia cells. Sommer's sector was no more affected than other portions of

the pyramidal layer. Lesions in the claustrum, putamen, caudate nucleus, epithalamus and medial hypothalamus were relatively scarce. The choroid plexuses were free from change except around larger vessels, where there were occasional sparse collections of lymphocytes. The olfactory bulbs were not available for study. The olfactory nerves and olfactory striae showed a few perivenous and parenchymal mononuclear cells. Neither the neurohypophysis nor the adenohypophysis revealed abnormalities.

In the midbrain, the substantia nigra was the seat of an extremely heavy concentration of nodules and diffusely distributed cells, together with perivenous collections of lymphocytes (fig. 8); the cell types were similar to those observed in the thalamus. Moderate numbers of ganglion cells had disappeared. The colliculi and the oculomotor and trochlear nuclei were involved to a slighter degree, and the red nucleus and the periaqueductal gray matter, least of all. In the rest of the brain stem, the most intense lesions were in the nuclei pontis. The nuclei in the floor of the fourth ventricle were also rather severely affected, in contrast to the relative sparing of the inferior olivary and arcuate nuclei. Here, as elsewhere in the brain, the white matter was virtually free from change.

Sections of the cerebellum indicated uniform distribution throughout the cerebellar folia. All the nodules were in the molecular layer and often pervaded the Bergmann layer and occasionally encroached on the granular layer. Discrete nodules, usually of greatest diameter at the base of the molecular layer, were most numerous either in regions from which the Purkinje cells had disappeared or in areas in which they were in a state of degeneration (fig. 9 *A* and *B*). More diffuse collections of cells, composed chiefly of the Hortega type, reached from the Bergmann layer to the pia; these, too, predominated in regions where Purkinje cells were damaged or absent (fig. 9 *C*). The dentate and roof nuclei were moderately affected, and, in contrast to the vessels in the rest of the cerebellum, the larger veins were frequently surrounded by lymphocytes.

The picture in the spinal cord was in agreement with that in the brain—the gray matter was involved and the white matter was relatively unaffected. At each spinal level examined, moderate numbers of neuronophagic nodules were sprinkled through the anterior horns (fig. 10 *A*), with a few in the posterior and lateral horns. As in other parts of the central nervous system, the cells in the perineuronal spaces were chiefly of the mononuclear variety. Large areas of the anterior horns were pervaded by these cells, and here and there one encountered neutrophils (fig. 10 *B* and *C*). An occasional gitter cell was observed, and sections stained with sudan III disclosed fat in only a few perivascular histiocytes. A few perivenous cuffs of lymphocytes extended from the anterior horns into the adjacent white matter. Moderate numbers of petechial hemorrhages were seen in the gray matter, especially in the region of the cervical portion of the cord, which on gross examination had an almost mushy appearance. Stains for myelin revealed nothing noteworthy.

*Changes in the Peripheral Nervous System.*—The gasserian ganglia, one dorsal root ganglion from the thoracic region and several spinal nerve roots were available for study. The only changes in the gasserian ganglia occurred in the surrounding dura, where a few petechial hemorrhages and a sparse sprinkling of lymphocytes and plasma cells were observed. A similar collection of cells was present in the epineurium of the dorsal root ganglion. The spinal nerve roots were without change.

*Changes in the Thoracic and Abdominal Viscera.*—Abnormalities of the thoracic and abdominal viscera were scanty. In no viscus were changes discerned in the vessel walls. The heart was normal except for an occasional small interstitial

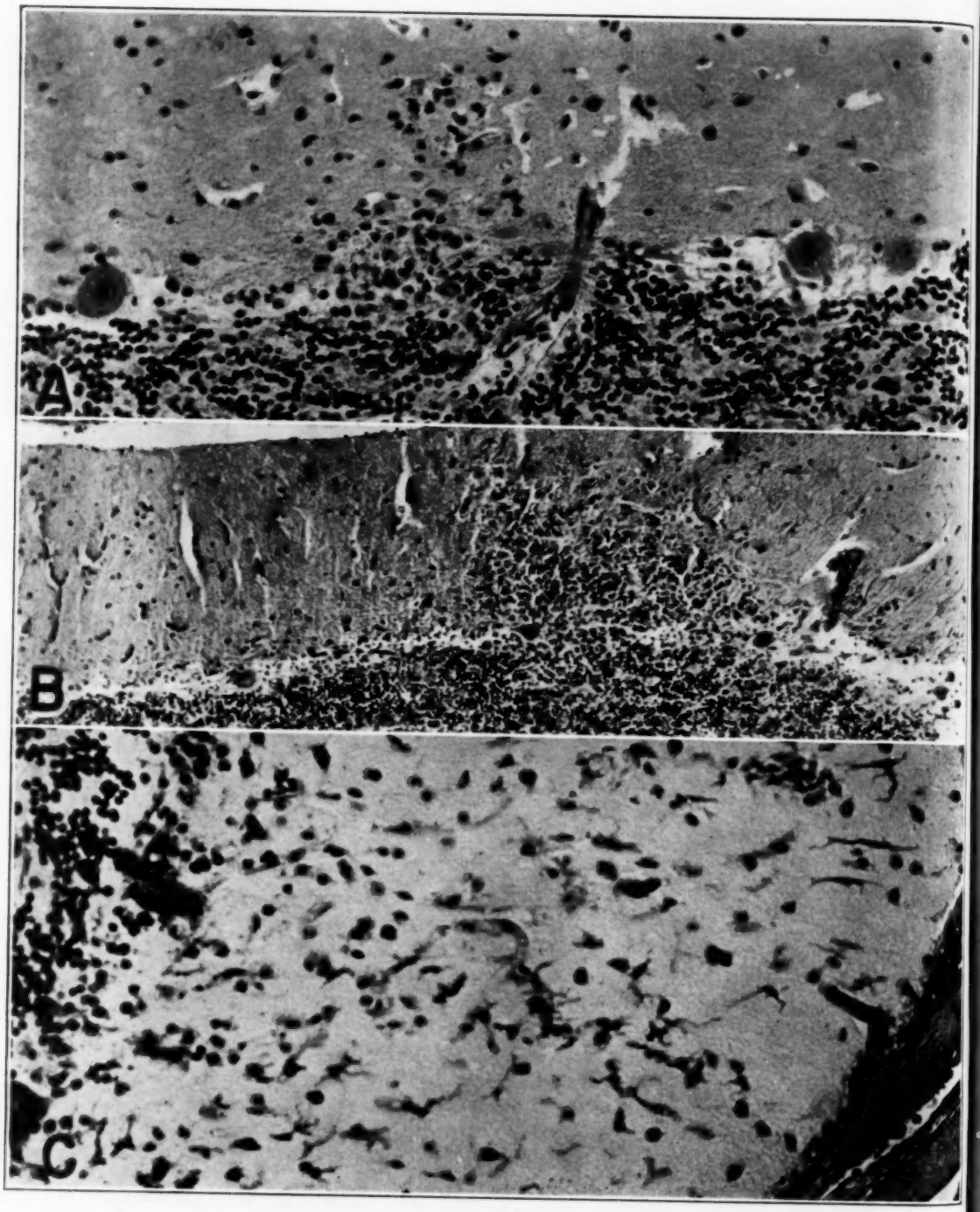



Figure 9

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collection of Anitschkow myocytes and lymphocytes. The lower lobe of each lung was the seat of acute bronchopneumonia. Both the liver and the spleen were congested, and their reticuloendothelial cells contained pigment, probably malarial. The cells of the adrenal cortex were depleted of lipid, and in the zona fasciculata a slight tubular degeneration, of the type described by Rich,[4] was observed.

## COMMENT

In this fulminant case of Japanese B encephalitis, one was struck, first, with the large number of neuronophagic nodules in all portions of the gray matter, and, second, with the more diffuse aggregations of cells which permeated the parenchyma. The great variation in the stages of evolution of the lesions permitted an evaluation of the nature of the cells participating in the reaction. It was apparent that the infiltrate around large veins was predominantly lymphocytic. The smaller the vessel, the greater the variety of perivascular cell forms; generally large mononuclear cells were in far greater abundance than the smaller, lymphocytic, forms. The same was true of the aggregates of cells surrounding degenerating or necrotic ganglion cells.

The smaller the vessel, the greater was the tendency of the cells to break through the membrana limitans gliae at focal points to invade the parenchyma. Where dense aggregations of cells permeated the parenchyma, a variety of cell forms was observed. If one may assume that these aggregations represent a far-flung invasion from perivascular and perineuronal spaces, spreading diffusely, or from the periphery of compact cell groups (nodules)—as would seem to be the case—one then is justified in concluding that most of the cells are migrants from the blood stream and from the adventitia, a view held by Maximow,[5] Michels and Globus[6] and others on the basis of observations on neurotropic virus diseases. The presence of "polyblastic" cells in nodules still confined to perivascular or perineuronal spaces and of cells of the

4. Rich, A. R.: Peculiar Type of Adrenal Cortical Damage Associated with Acute Infections, and Its Possible Relation to Circulatory Collapse, Bull. Johns Hopkins Hosp. **74**:1-15, 1944.

5. Maximow, A.: Untersuchungen über Blut und Bindegewebe, Arch. f. mikr. Anat. **73**:444-561, 1909; **76**:1-113, 1910; **96**:494-527, 1922.

6. Michels, N. A., and Globus, J. H.: The So-Called Small Round Cell Infiltrations: I. Polio-Encephalitis and Acute Epidemic Encephalitis, Arch. Path. **4**:692-731 (Nov.) 1927.

Fig. 9.—Representative lesions in the cerebellum. In *A* (× 280; AIP neg. 94484), a nodule composed of pleomorphic mononuclear cells is present at the base of the molecular layer. Presumably, it is forming at the site of a destroyed Purkinje cell. Hematoxylin and eosin stain. In *B* (× 110; AIP neg. 92621), a nodule of greater scope is illustrated. Hematoxylin and eosin stain. In *C* (× 350; AIP neg. 93073), a diffuse aggregate of cells permeates the entire width of the molecular layer. Some of the cells are in the category of mononuclear cells, but the majority have the characteristics of Hortega cells. One of the Purkinje cells appears to be disintegrated. Cresyl violet stain.

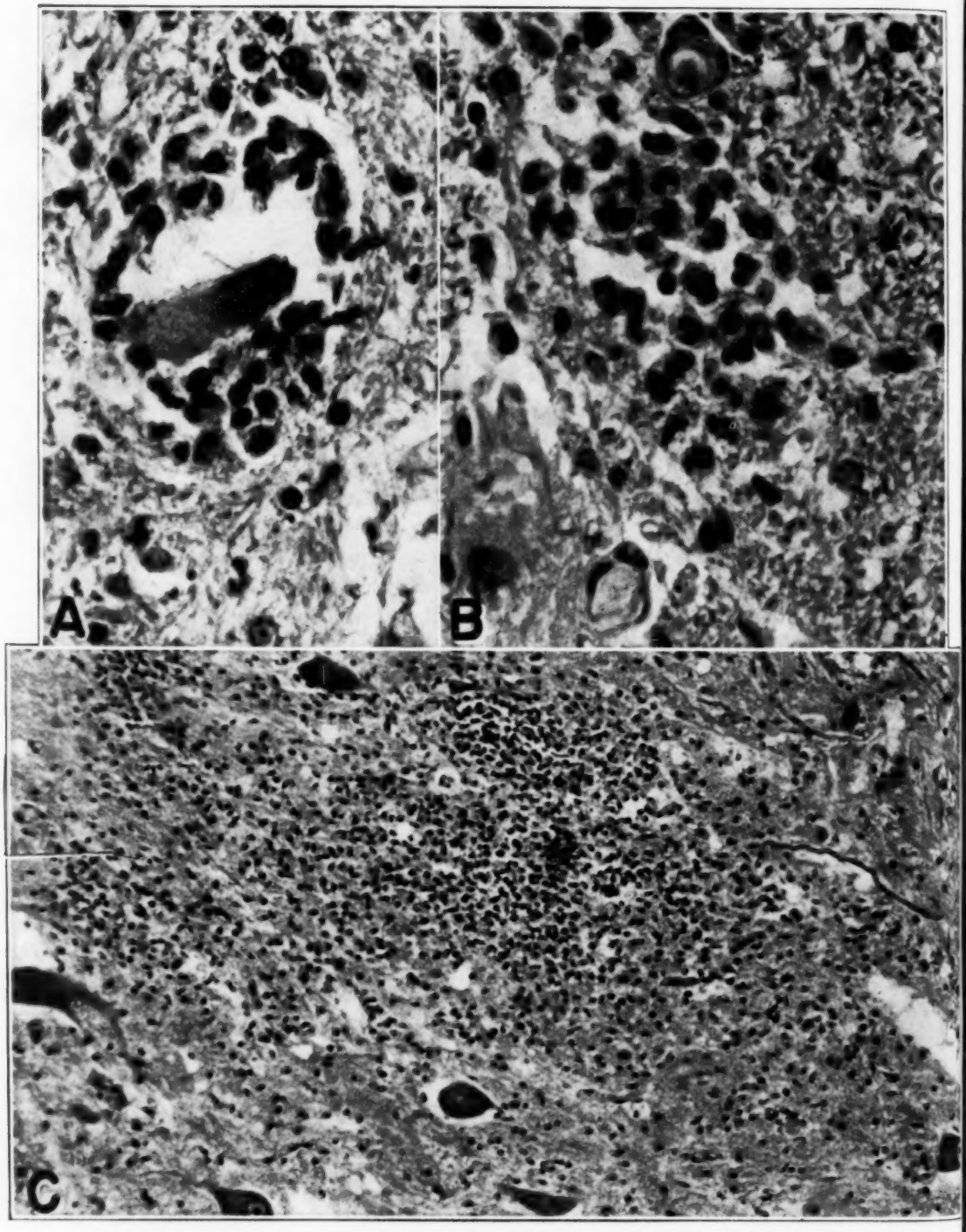


Figure 10

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same appearance in the front lines of the parenchymal invasion is in disagreement with the view of Hurst [7] that the great majority of "polyblastic" forms in poliomyelitis—in which the problem of cytogenesis, in our opinion, is virtually the same as that in Japanese B encephalitis —are mobilized Hortega cells. In our case there is no denying the presence of proliferated Hortega cells in some regions of the brain, notably the cerebellum, where they took the form of diffuse aggregates, but it is doubtful whether they were present to any appreciable degree in the dense parenchymal morass. One wonders how frequently in the case of other virus encephalitides the application of "glial nodule" to nodular lesions and of "glial proliferation" to diffuse lesions is ill advised.

In the differential diagnosis of Japanese B encephalitis, as of the other arthropod-borne encephalitides, only two diseases need consideration: lethargic (von Economo) encephalitis and poliomyelitis. The pathologic changes in the substantia nigra in our case were indistinguishable from those of lethargic encephalitis, but the over-all pictures of the two diseases were different. In lethargic encephalitis the lesions in the cerebral cortex seldom, if ever, take the form of nodules; they tend to be more concentrated in the upper portion of the midbrain, the posterior hypothalamus and the subthalamus; they do not affect in so massive a fashion the thalamus, and they often fail to involve appreciably the spinal cord. The lesions in the spinal cord in our case were virtually the same as those in poliomyelitis, except that at some levels they were more spotty than is ordinarily encountered in poliomyelitis. In other ways the two diseases are distinct, there being in poliomyelitis absence of involvement of the cerebral cortex except in the region of the precentral gyrus, sparing of the molecular and the Purkinje cell layers of the cerebellum, though the cerebellar nuclei are affected, and an attack on the spinal root ganglia.[8]

The types of lesions and their distribution in our case were essentially the same as observed by Takeno [9] in 8 cases of Japanese B encepha-

7. Hurst, E. W.: The Histology of Experimental Poliomyelitis, J. Path. & Bact. **32**:457-477, 1929.

8. Sabin, A. B.: Pathology and Pathogenesis of Human Poliomyelitis, J. A. M. A. **120**:506-511 (Oct. 17) 1942.

9. Takeno, K.: Histopathologie der japanischen epidemischen Encephalitis, Okayama Igakkai-Zasshi **44**:1459-1482, 1932.

Fig. 10.—Sections from the anterior horns of the spinal cord. In *A* (× 750; AIP neg. 94446), the perineuronal space surrounding a necrotic ganglion cell is crowded with mononuclear cells. An occasional cell has the features of the oligodendrocyte. In B (× 810; AIP neg. 94442), a collection of cells at the periphery of the anterior horn is shown. Some with bilobed and trilobed nuclei are neutrophils; the others are of the mononuclear variety. In *C* (× 160; AIP neg. 94448) is illustrated a densely pervaded area in the anterior horn from which ganglion cells have disappeared. Hematoxylin and eosin stain.

litis occurring in Japan between 1924 and 1929. In all instances the thalamus and the substantia nigra were the structures most severely affected. In cases of short survival infiltration of leukocytes into the parenchyma was an outstanding feature. The inferior olivary nucleus was more often affected in his cases than in our case. Takeno made no mention of the spinal cord. Pathologic changes closely resembling those described by us were observed also in 2 cases of Japanese B encephalitis in Okinawa, from which representative blocks of tissue were submitted to the Army Institute of Pathology by Lieut. Comdr. H. M. Zimmerman [10] of the United States Navy. In 1 case in which the duration of illness was twenty-three days, the thalamus was the seat of widespread and severe malacia, and in the other, in which death occurred in three days, the thalamus contained, in addition to nodules and a diffuse infiltrate, numerous relatively large focal necroses, in which only a spongy network of parenchyma remained. The substantia nigra, cerebellum and spinal cord in these cases were slightly less affected than in our case, while the lesions in the cerebral cortex were of the same severity. It would appear, therefore, that ours is a representative case of Japanese B encephalitis.

## CONCLUSIONS

A detailed study of a case of Japanese B encephalitis occurring in a member of the United States armed forces in Okinawa, in which the duration of the illness was approximately ten days, revealed a striking involvement of the gray matter of the central nervous system, relative sparing of the white matter and absence of change in the peripheral nerves and in the thoracic and abdominal viscera. The structures most severely affected were the thalamus, the substantia nigra, the nucleus basalis and the anterior horns of the spinal cord, where the lesions tended to be confluent, and the cerebral cortex and the cerebellum, where they generally were discrete. The presence of neuronophagic nodules in all portions of the gray matter was taken as compelling evidence of damage to ganglion cells by the virus. These nodules, generally spoken of as "glial," we regard as composed of hematogenous and adventitial mononuclear cells.

A review of cases of Japanese B encephalitis previously recorded in the literature and of 2 additional ones that came under our observation, makes it evident that the case described by us is a representative one.

The involvement of the gray matter of all regions of the cerebral cortex and of the molecular and Purkinje cell layers of the cerebellum in cases of Japanese B encephalitis serves to differentiate the topographic distribution of the lesions in this disease from that of the lesions in poliomyelitis.

10. Zimmerman, H. M.: Pathology of Japanese B Encephalitis, Am. J. Path. **22**:965-991, 1946.

# ELECTROCEREBRAL SHOCK THERAPY

## A Reconsideration of Former Contraindications

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WITH the advent of new procedures and drugs in the field of applied medicine, there may be a tendency to an overenthusiastic application of these methods, with the result that failures occur in frequently unlooked-for, untoward effects. The result is a swing in the opposite direction of conservatism and overcautiousness, and many patients who would benefit from a new form of treatment are refused it. The severe convulsive reactions encountered with metrazol shock and the early inexperience with methods of overcoming or subduing complications produced a timidity with respect to contraindications which carried over to electric shock therapy.

It is not at all unlikely that the present seemingly drastic and terrifying forms of shock therapy will be superseded by some gentle chemical process based on a rational biochemical-physiologic understanding of the why and wherefore of the salutary response of certain psychoses to shock therapy. Until such time, however, the present day advantages to be obtained from shock therapy should be utilized in properly selected cases.

A sufficiently large body of literature and experience has accumulated to give one information in reevaluating the indications for and contraindications to cerebral electric shock treatment, a complete review of which is much too voluminous to incorporate in this paper. Some of the reviews with respect to the indications for, results of, and complications attending, electric shock treatment may be found in the works of various authors.[1]

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From the Philadelphia Psychiatric Hospital.

Read before the Section of Nervous and Mental Diseases, at the Ninety-Fifth Annual Session of the American Medical Association, San Francisco, July 4, 1946.

1. (*a*) Almansi, R., and Impastato, D. J.: Electrically Induced Convulsions in the Treatment of Mental Diseases, New York State J. Med. **40**:1315-1316, 1940. (*b*) Bennett, A. E.: An Evaluation of the Shock Therapies, Psychiatric Quart. **19**:465-477, 1945; (*c*) Unusual Organic Complicating Factors in Convulsive Shock Therapy, Bull. Menninger Clin. **8**:71-73, 1944. (*d*) Bingel, A.: Ueber die psychischen und chirurgischen Komplikationen des Elektrokrampfes, Allg. Ztschr. f. Psychiat. **115**:325-343, 1940. (*e*) Darling, H. F.: Prevention of Fatality and Fracture During Electric Coma Therapy, J. Nerv. & Ment. Dis. **100**:70-72, 1944.

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The contraindications which, until recently, have been considered an unquestioned barrier to convulsive shock treatment are acute infections and febrile diseases, cardiovascular disease, renal disease, advanced age, bone atrophy in long-bedridden patients, pronounced spinal curvature and osteoarthritis, severe thyrotoxicosis, malignant tumors, thrombo-

(*f*) Davidoff, E., and Raffaele, A.: Electric Shock Therapy in Involutional Psychoses, ibid. **99**:397-405, 1944. (*g*) Ebaugh, F. G.; Barnacle, C. H., and Neubuerger, K. T.: Fatalities Following Electric Convulsive Therapy: Report of Two Cases with Autopsy, Arch. Neurol. & Psychiat. **49**:107-117 (Jan.) 1943. (*h*) Epstein, J.: Electric Shock Therapy in the Psychoses: Study of One Hundred Cases, J. Nerv. & Ment. Dis. **98**:115-129, 1943. (*i*) Gralnick, A.: Fatalities Associated with Electric Shock Treatment of Psychoses: Report of Two Cases with Autopsy Observation in One of Them, Arch. Neurol. & Psychiat. **51**:397-402 (April) 1944; (*j*) A Three-Year Survey of Electroshock Therapy: Report of Two Hundred and Seventy-Six Cases; Comparative Value of Insulin-Coma Therapy, Am. J. Psychiat. **102**:583-593, 1946. (*k*) Hemphill, R. E.: Electrical Convulsion Therapy: Clinical Observations, Lancet **2**:152-154, 1942. (*l*) Huddleson, J. H.: Complications in Electric Shock Therapy, Am. J. Psychiat. **102**:594-598, 1946. (*m*) Impastato, D. J., and Almansi, R.: Study of Over Two Thousand Cases of Electrofit-Treated Patients, New York State J. Med. **43**:2057-2063, 1943; (*n*) The Electrofit in the Treatment of Mental Disease, J. Nerv. & Ment. Dis. **96**:395-409, 1942. (*o*) Impastato, D. J.; Frosch, J.; Almansi, R., and Wortis, S. B.: The Electrofit in Depression: Comparison of Hospital and Privately Treated Patients, New York State J. Med. **45**:179-185, 1945. (*p*) Jetter, W. W.: Fatal Circulatory Failure Caused by Electric Shock Therapy, Arch. Neurol. & Psychiat. **51**:557-563 (June) 1944. (*q*) Jones, G. L., in symposium on Complications of and Contraindications to Electric Shock Therapy, ibid. **49**:788-789 (May) 1943. (*r*) Kalinowsky, L. B.: Experience with Electric Convulsive Therapy in Various Types of Psychiatric Patients, Bull. New York Acad. Med. **20**:485-494, 1944. (*s*) Kalinowsky, L. B.; Barrera, S. E., and Horwitz, W. A.: Electric Convulsive Therapy of the Psychoneuroses, Arch. Neurol. & Psychiat. **52**:498-504 (Dec.) 1944. (*t*) Kalinowsky, L. B., and Worthing, H. J.: Results with Electric Convulsive Treatment in Two Hundred Cases of Schizophrenia, Psychiatric Quart. **17**:144-153, 1943. (*u*) Kalinowsky, L. B., and Hoch, P. H.: Shock Treatments and Other Somatic Procedures in Psychiatry, New York, Grune & Stratton, Inc., 1946, p. 294. (*v*) Kalinowsky, L. B.: Electric Convulsive Therapy, with Emphasis on Importance of Adequate Treatment, Arch. Neurol. & Psychiat. **50**:652-660 (Dec.) 1943; (*w*) Organic Psychotic Syndromes Occurring During Electric Convulsive Therapy, ibid. **53**:269-273 (April) 1945. (*x*) Kline, E. M., and Fetterman, J. L.: Electrocardiograph Changes Following Electrically Induced Convulsions, Am. Heart J. **24**:665-670, 1942. (*y*) Kolb, L., and Vogel, V. H.: The Use of Shock Therapy in Three Hundred and Five Mental Hospitals, Am. J. Psychiat. **99**:90-100, 1942. (*z*) Lewis, N. D.: The Present Status of Shock Therapy of Mental Disorders, Bull. New York Acad. Med. **19**:227-244, 1943. (*a'*) Lowinger, L., and Huddleson, J. H.: Outcome in Dementia Praecox Under Electric Shock Therapy as Related to Mode of Onset and to Number of Convulsions Induced, J. Nerv. & Ment. Dis. **102**:243-246, 1945. (*b'*) Levy, N. A.; Serota, H. M., and Grinker, R. R.: Disturbance of Brain Function Following Convulsive Shock Therapy, Arch. Neurol. & Psychiat. **47**:1009-1029 (June) 1942. (*c'*) Meyer, A., and

*(Footnote continued on next page)*

phlebitis, advanced general arteriosclerosis, organic disturbances of the central nervous system and tuberculosis, according to Cook [2] "the most important absolute contraindication . . . even if only latent."

Some of these contraindications can now, in the light of experience and judgment, be eliminated from this category and greater freedom of use of the method be permitted. This applies with respect to cardiovascular diseases with and without hypertension; advanced age; latent, arrested or even active tuberculosis; some cases of generalized arteriosclerosis in which a differential diagnosis between cerebral vascular involvement and involutional psychosis has not been established; organic disease of the central nervous system, such as dementia paralytica; spinal curvatures, and osteoarthritis.

The present reconsideration of some of the heretofore believed contraindications to electric convulsive treatment is based on a review of the cases of 1,596 intramural and 585 extramural patients treated at the Philadelphia Psychiatric Hospital from Nov. 4, 1940 to May 5, 1946. These 2,181 patients received electric shock treatment either alone or in conjunction with insulin. They received a total of 20,645 convulsive treatments, or an average of 9.5 treatments per patient.

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Teare, D.: Cerebral Fat Embolism After Electrical Convulsion Therapy, Brit. M. J. **2**:42-44, 1945. (*d'*) Myerson, A.: Further Experience with Electric-Shock Therapy in Mental Disease, New England J. Med. **227**:403-407, 1942; (*e'*) Prolonged Cases of Grief Reaction Treated by Electric Shock, ibid **230**:255-256, 1944. (*f'*) Napier, F. J.: Death from Electrical Convulsion Therapy, J. Ment. Sc. **90**:875-878, 1944. (*g'*) Norman, J., and Worthington, R. V.: Electric Shock Treatment in One Hundred Cases, Dis. Nerv. System **5**:236-242, 1944. (*h'*) Olsen, C. W.: An Unusual Reaction to Electroshock Treatment, Unilateral Convulsion and Transient Hemiplegia, Bull. Los Angeles Neurol. Soc. **9**:171-173, 1944. (*i'*) Pacella, B. L.: Sequelae and Complications of Convulsive Shock Therapy, Bull. New York Acad. Med. **20**:575-587, 1944. (*j'*) Pacella, B. L.; Barrera, S. E., and Kalinowsky, L. B.: Variations in Electroencephalogram Associated with Electric Shock Therapy of Patients with Mental Disorders, Arch. Neurol. & Psychiat. **47**:367-384 (March) 1942. (*k'*) Rennie, T. A.: Present Status of Shock Therapy, Psychiatry **6**:127-137, 1943. (*l'*) Rosen, S. R.; Secunda, L., and Finley, K.: The Conservative Approach to the Use of Shock Therapy in Mental Illness, Psychiatric Quart. **17**:617-641, 1943. (*m'*) Samuel, E.: Complications Arising During Electric Convulsive Therapy, J. Ment. Sc. **89**:81-84, 1943. (*n'*) Smith, L. H.; Hastings, D. W., and Hughes, J.: Immediate and Follow-up Results of Electroshock Therapy, Am. J. Psychiat. **100**:351-354, 1943. (*o'*) Taylor, H. J., Jr., in symposium on Complications of and Contraindications to Electric Shock Therapy, Arch. Neurol. & Psychiat. **49**:789-790 (May) 1943. (*p'*) Taylor, J. H.: A Further Report on the Use of Shock Therapy: Results in One Thousand Three Hundred and Two Cases, Dis. Nerv. System **5**:56, 1944. (*q'*) Woolley, L. F.: Immediate Circulatory and Respiratory Effects of Convulsive Shock, J. Nerv. & Ment. Dis. **100**:1-23, 1944. (*r'*) Worthing, H. J., and Kalinowsky, L. B.: The Question of Vertebral Fractures in Convulsive Therapy and in Epilepsy, Am. J. Psychiat. **98**:533-537, 1942.

2. Cook, L. C.: Recent Progress in Psychiatry: Convulsion Therapy, J. Ment. Sc. **90**:435-464, 1944.

Eight of the 9 cases cited here are representative of cases in which some of the previously mentioned contraindications to electric shock treatment existed, and in which the question of weighing the advantages and likelihood of recovery with the use of shock therapy against the potential dangers arose as a problem of the hospital's versus the attending physician's responsibility. Each patient was studied carefully before shock treatment was decided on; precautions were observed at the time of treatment, and a thorough physical check-up was made before further treatments were given.

The results obtained in the treatment of the 1,596 intramural patients were as follows: patients recovered, 552; patients improved, 838; patients unimproved, 201; patients without psychoses, 2; deaths, 2; suicide, 1. The diagnoses for these patients are found in table 1.

TABLE 1.—*Psychiatric Diagnoses for 1,596 Intramural Patients Treated with Electric Shock*

| | No. of Patients |
|---|---|
| Manic-depressive psychosis—all types | 497 |
| Schizophrenia—all types | 478 |
| Involutional psychosis | 273 |
| Psychoneuroses | 252 |
| Paranoid states | 29 |
| Psychosis with cerebral arteriosclerosis | 14 |
| Psychosis with alcoholism | 5 |
| Senile psychosis | 4 |
| Psychosis with syphilitic meningoencephalitis | 4 |
| Mental deficiency without psychosis | 3 |
| Dementia paralytica | 2 |
| Psychosis with cardiovascular disease | 2 |
| Behavior problem | 2 |
| Adult maladjustment | 2 |
| Toxic exhaustion state | 1 |
| Toxic psychosis—thyroid | 1 |
| Psychosis due to lead poisoning | 1 |
| Psychosis with meningovascular syphilis | 1 |
| Psychosis with organic cerebral disease | 1 |
| Psychosis with drug addiction | 1 |
| Epilepsy with equivalent states | 1 |
| Traumatic psychoneurosis | 1 |
| Psychopathic personality with pathologic emotionality | 1 |
| Psychopathic personality with pathologic sexuality | 1 |
| Undiagnosed psychoses | 19 |
| Total | 1,596 |

It will be noted that organic disease of the brain is well represented in the list of disorders, with cerebral arteriosclerosis and syphilis of the brain predominating. Cerebral arteriosclerosis was a complicating factor in most cases of depression in which electric shock was salutary. Syphilis of the brain, in its various forms, cannot be considered a valid contraindication, the only report to the contrary being that by Heilbrunn and Feldman,[3] who observed cardiovascular and respiratory failure in patients whose condition was diagnosed as psychosis with syphilitic meningoencephalitis.

Advanced age is becoming a somewhat relative term, and as a contraindication to electric shock treatment it is not a deterrent per se,

3. Heilbrunn, G., and Feldman, P.: Electric Shock Treatment in General Paresis, Am. J. Psychiat. **99**:702-705, 1943.

as is already manifest from the reports of several observers.[4] Of the intramural patients, 39 were over 60, 15 over 65 and 2 over 70, years of age. The oldest patient was 74. Incidentally, the youngest patient in this series was 14. Eighteen patients had general arteriosclerosis and 190 patients had hypertension. No complications followed treatment. Evans [5] reported 9 patients with systolic pressure over 200 mm. of mercury to whom electroshock treatment was given without complications. In some patients the initial high pressure falls after shock treatment. Kennedy and Wiesel [6] reported a striking example of this.

The most frequently occurring organic disorder in this series of patients was myocardial disease, in its various forms. Table 2 summarizes the types of cardiac disease encountered.[7]

TABLE 2.—*Forms of Myocardial Disease Encountered in Series of 1,596 Patients Treated by Electric Shock* *

| | No. of Patients |
|---|---|
| Number of patients with electrocardiographic studies | 1,756 |
| Diseases of the myocardium | 57 |
| Rheumatic heart disease | 25 |
| Hypertensive cardiovascular disease | 21 |
| Coronary arteriosclerosis | 16 |
| Coronary artery disease | 9 |
| Myocardial infarction | 9 |
| Mitral stenosis | 7 |
| Aortic stenosis | 2 |
| Branch bundle block | 8 |
| Auricular-ventricular block | 5 |
| Endocarditis | 1 |
| Other cardiac disturbances | 78 |
| Total number of patients with heart disorders | 238 |

* A more detailed analysis of these conditions and of the electrocardiographic studies is now in preparation.

The following case reports illustrate some of the serious cardiac disturbances, as found on clinical and electrocardiographic study, which were not adversely affected by electric shock treatment, with 1 exception, to be reported later.

CASE 1.—F. A., a married woman aged 59, was first examined on March 2, 1944 and admitted to the hospital on March 13. Six months before there developed anxiety, light-headedness, lack of interest, insomnia, loss of appetite, loss of confidence, depression, lapses of memory and numerous somatic complaints. In December 1943 she was in another psychiatric institution for several weeks, where

4. Evans, V. L.: Convulsive Shock Therapy in Elderly Patients: Risks and Results, Am. J. Psychiat. **99**:531-533, 1943. Mayer-Gross, W.: Electric Convulsion Treatment in Patients Over 60, J. Ment. Sc. **91**:101-103, 1945.

5. Evans, V. L.: Electroconvulsive Shock Therapy and Cardiovascular Disease, Ann. Int. Med. **22**:692-695, 1945.

6. Kennedy, F., and Wiesel, B.: A Report on the Results of Electric Shock Treatment on Mental and Emotional Symptoms, New York State J. Med. **42**:1663-1668, 1942.

7. The cardiologic and electrocardiographic studies were made by Dr. Benjamin Gouley, Philadelphia.

shock treatment was refused because of hypertension. After study the diagnosis of involutional psychosis with depression was made. The systolic blood pressure varied from 180 to 210 mm. of mercury; the diastolic pressure was 110 mm. The electrocardiographic report prior to treatment follows: "The cardiac rate is 68 per minute; the PR interval is 0.16 second; the P waves are split in leads I and III; the QRS complexes are slightly shaded, with left axis deviation. A very small upward deflection is shown in lead IV; the T waves in leads I and II are inverted. The electrocardiographic diagnosis is that of a pathologic condition, the tracing suggesting mild hypertensive cardiovascular disease, dilatation and healed infarction at the apex. The patient should be treated with caution."

Responsibility for treatment was placed by the hospital on the attending physician. After four electric shock treatments with full convulsive reactions, the patient was somewhat confused, and no improvement was observed except in her appetite. At this point the staff physician expressed the belief that her psychosis was due to cerebral arteriosclerosis and suggested termination of treatment. However, shock therapy was continued, and after the sixth treatment striking improvement was apparent. Eight treatments were given, and the patient was discharged one month after admission as recovered. To date, she has maintained complete recovery. The electrocardiogram on April 12, 1944, following the course of treatment, showed no change.

Case 2.—M. H., a married woman aged 63, was first examined April 14, 1944 and admitted to the hospital April 17. For six years she had shown an increasing degree of mental disturbance, having had several "nervous breakdowns" during the first two of these six years and for the past four years manifesting disinterestedness, a feeling of inadequacy, inertia, numerous somatic complaints, ideas of persecution, irritability, depression and ideas of self destruction. There had been a loss of 50 pounds (22.7 Kg.) in weight, and she was emaciated and dehydrated on admission to the hospital. The diagnosis of manic-depressive psychosis, depressed type, was made. The blood pressure showed systolic variations from 116 to 140 mm. of mercury and a diastolic pressure of 70 to 76 mm. Electrocardiographic studies (April 17) were reported as follows: "The cardiac rate is 75 per minute; the PR interval is 0.16 second; the P waves are of rather low amplitude but otherwise normal; the interval of the QRS complexes is 0.06 second. There is a small initial upward deflection in the direct lead; the T waves are biphasic in lead I, inverted in leads II and III and flat to inverted in lead IV, with a slight elevation of the S-T interval in leads I and II. The electrocardiographic diagnosis is as follows: The tracing suggests a pathologic condition, with widespread myocardial degeneration, due most likely to sclerotic disease of the coronary arteries. The patient should be treated with caution."

Treatment with electric shock was begun, and after induction of the third shock, and again after the termination of twelve shock treatments, the electrocardiographic tracings were repeated. The report on the electrocardiographic studies on June 3, 1944 follows: The cardiac rate is 78 per minute; the PR interval in lead I appears to be 0.18 second; in lead II, and also in lead III, it is more readily observed to be 0.28 second. The initial major deflection in lead IV is missing."

At the time of discharge, on June 3, 1944, the patient had shown notable physical improvement, and, although her psychomotor activity was still subdued, the depression and numerous somatic references had disappeared. Subsequent follow-up observations showed a gain of 15 pounds (6.8 Kg.) in weight and improved adaptability to social and household life.

CASE 3.—F. L. S., an unmarried woman aged 62, was first examined Dec. 20, 1944. During May 1944, after treatment for corneal ulcers, she experienced the fear of becoming blind. She then manifested anxiety, seclusiveness, refusal to eat, wringing of the hands, picking at the skin, ideas of unworthiness, self accusation, somatic delusions and agitation. She was admitted to a hospital for mental diseases in Kansas City, Mo., in August 1944, where she received fifteen metrazol shock treatments. Although some improvement took place, at the time of her discharge, in November, she was still mildly agitated. When examined in December, she presented the clinical picture of agitated involutional melancholia. Physical exami-

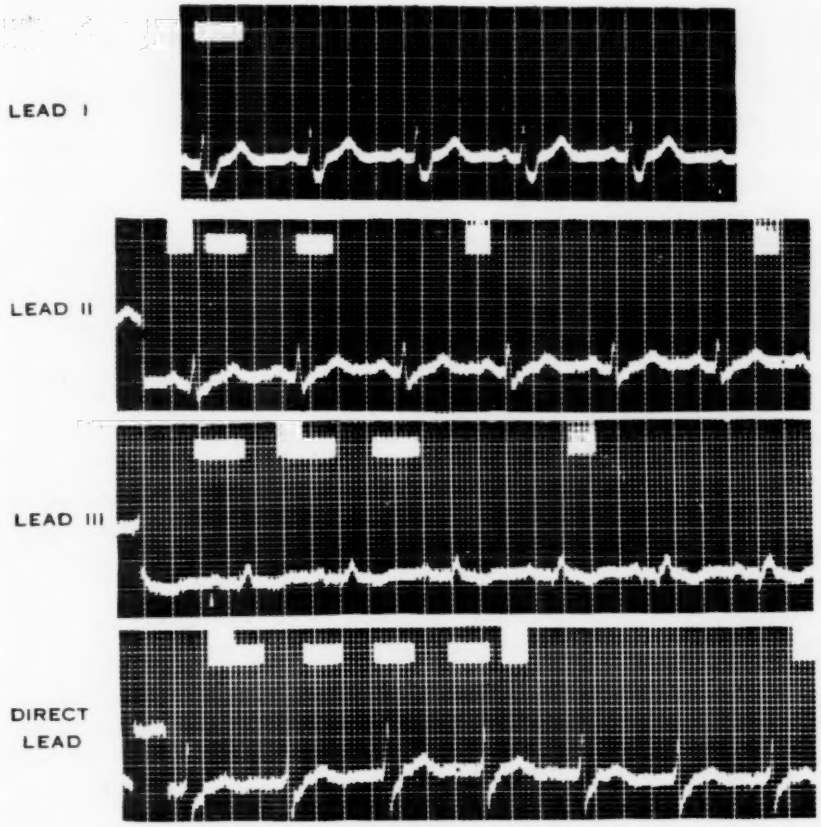


Fig. 1 (case 3).—Electrocardiographic tracing.

nation showed evidence of generalized arteriosclerosis, enlargement of the heart, presystolic and systolic murmurs at the apex and a blood pressure of 175 systolic and 80 diastolic. The electrocardiographic study (December 21) was reported as follows: "The cardiac rate is 78 per minute, with normal sinus rhythm; the PR interval is 0.20 second (with normal top); the P waves show splitting in lead II; the QRS complexes indicate a right bundle branch block; the T waves are upright in leads I, II and IV and inverted in lead III. The electrocardiographic diagnosis is right bundle branch block; delayed A-V conduction time, and cardiac lesions associated with enlargement of and strain on the right ventricle (fig. 1)."

The patient began to show a favorable response after the third shock treatment, and after the seventh electric shock she was slightly euphoric, in contrast to the previous depression. The electrocardiogram on Feb. 5, 1945 was reported as follows: "The interval of the QRS complexes is 0.14 second, indicating right bundle branch block. The tracing remains substantially unchanged. The T wave in lead IV is now inverted, instead of upright." The patient was discharged as recovered.

CASE 4.—S. M., a single man aged 32, was first examined on Feb. 13, 1945 and admitted to the hospital on February 22. Two years before, he had enlisted in the Navy, knowing that he had heart disease, and was subsequently discharged when the cardiac condition was discovered. He stated the belief that he had been "fraudulently discharged." After this there developed ideas of reference (that people were calling him a slacker), suspiciousness and ideas of persecution (of being trailed by the F. B. I. and the Navy intelligence). He became slovenly in attire and abusive and assaultive to his part time common law wife. He was employed up to four weeks prior to admission. He then became greatly agitated and panicky. After psychiatric study, the provisional diagnoses of (1) paranoid psychosis with acute panic state and (2) schizophrenia, paranoid type, were entertained. The referring physician, a cardiologist, stated that the patient had "old rheumatic mitral valvular disease, perfectly compensated." An electrocardiogram (February 26) was reported as follows: "The cardiac rate is 93 per minute; the PR interval is 0.16 second; the P waves are rather prominent in leads II and III; the QRS complexes are 0.09 second and somewhat widened; the S waves are deep in lead II. Left axis deviation is present; the T waves are upright in lead I, biphasic in lead II, inverted in lead III and upright in lead IV; the S-T interval is elevated in lead IV. The electrocardiographic diagnosis is as follows: The tracing suggests hypertensive cardiovascular disease, with myocardial degeneration in the posterior wall of the left ventricle. There is undoubtedly a pathologic condition of the heart of a nature which is serious for a person of the patient's age (32 years). I believe that he has serious disease of the coronary arteries."

"NOTE.—Clinical examination changes somewhat the concept of this case. There is a murmur of aortic regurgitation. The left ventricle is moderately enlarged. There is a presystolic mitral murmur (mitral valvulitis? Austin Flint murmur?). In view of the negative serologic reactions, I suspect that rheumatic disease is the pathologic basis. The electrocardiogram is that of myocardial degeneration. The blood pressure is 135 systolic and 70 diastolic."

After having received his third electric shock treatment, the patient became disorderly and abusive and made an unsuccessful attempt to escape. He was greatly confused and completely lacking in insight. After sixteen days of residence, because of his intractable behavior, he was transferred to another institution. Here he received three more electric shock treatments and made a remarkably rapid recovery, with disappearance of the confusion, agitation and ideas of reference. He was discharged as improved, and recent follow-up observation reveals that he has made a satisfactory social adjustment at his prepsychotic level. Examination by his cardiologist showed no change in the heart.

One of the patients with bundle branch block, a condition which is supposedly fixed in its electrocardiographic and clinical aspects, showed improvement in the electroencephalogram for two weeks during electric shock treatment, with subsequent reversion to the original pattern.

CASE 5.—One of the deaths in this series was that of S. M., a woman aged 52, who had an involutional depression with distressing agitation. She was known to have had several myocardial infarctions due to disease of the coronary arteries, both from the history and from electrocardiographic studies, and she also had diabetes mellitus. Her blood sugar varied from 130 to 210 mg. per hundred cubic centimeters; despite the risk involved, it was felt advisable, in view of the serious mental disturbance which the patient presented, to give electric shock therapy. She received three treatments, to the first two of which there were a petit mal response and no response, respectively. Only the third treatment resulted in a full convulsive reaction. Substernal pain developed after the third treatment, and an electrocardiogram revealed evidence of a fresh infarction. The patient did moderately well and then died suddenly four weeks after the third treatment. Permission for autopsy was not granted.

In reviewing the results for the 238 patients who presented cardiac disorders of mild, moderate and relatively severe forms, it was felt that, with the exception just cited, they withstood electric shock treatment well. I concur with Pacella,[11] who said:

> . . . it has been our experience and the experience of many others that individuals with varying degrees of cardiovascular disease, with or without abnormalities in the electrocardiogram, ordinarily tolerate convulsions well, providing the disease is not too acute or severe.

Sixty-three patients in this series showed some form of endocrine disturbance, as seen in the following tabulation.

| Endocrine Disorders | No. of Patients |
|---|---|
| Enlarged thyroid | 26 |
| Diabetes mellitus | 12 |
| Pituitary disturbance | 10 |
| Hyperthyroidism | 5 |
| Hypothyroidism | 5 |
| Ovarian dysfunction | 2 |
| Hypogonadism | 2 |
| Adenoma of thyroid with toxicity | 1 |

The patients with diabetes mellitus were stabilized by dietary measures alone or in conjunction with insulin before treatment was instituted. This condition in no way interfered with or complicated treatment. Despite the absence of complications in the cases of hyperthyroidism and the case of adenoma of the thyroid with toxicity, I should not advocate electric convulsive therapy in a case of pronounced exophthalmic goiter without first giving proper attention to the altered constitutional state and the thyroid gland itself.

It is questionable whether complications have ever arisen during convulsive shock treatment as the result of disturbances of the bony structures and joints themselves. Forty patients, as indicated in the following tabulation, had some type of skeletal disorder. No fractures

| Skeletal Disorders | No. of Patients |
|---|---|
| Scoliosis | 14 |
| Kyphosis | 12 |
| Kyphoscoliosis | 6 |
| Arthritis (chronic) | 6 |
| Osteomyelitis | 1 |
| Tuberculosis of the shoulder | 1 |

or dislocations occurred in this group, and none of the original conditions was worsened. Fractures or dislocations can be minimized or completely avoided by the present method [8] of proper splinting of the torso, limbs and head, using four or five attendants strategically placed. If fracture should occur, it can mend while the patient is improving mentally.

CASE 6.—F. B., an unmarried woman aged 57, was first examined March 10, 1945 and admitted to the hospital April 6, 1945. Approximately two years before admission a state of anxiety developed and she quit her job as bookkeeper, which she had held with the same concern for thirty-three years. For several months she was bedridden, displaying inertia, self-accusatory ideas and severe insomnia. Her illness had become more pronounced during the last two months and was characterized by hyperreligiosity, ideas of sinfulness and unworthiness, depression, numerous somatic complaints, obsessive thoughts of an obscene, tormenting, distressing nature, restlessness, and at times frenzied agitation. A diagnosis of involutional psychosis was made.

Physical examination showed a slight woman, weighing 91½ pounds (41.5 Kg.), who had pronounced scoliosis and kyphosis. This had been present since childhood. The heart sounds were irregular, with occasional premature systoles. The blood pressure was 110 systolic and 80 diastolic; the pulse rate was 90. The electrocardiographic report (April 7) was as follows: "The P waves show splitting in leads II and III; the T waves are inverted in leads II and III; the tracing is abnormal. Treatment should be withheld. It is suspected that this patient has coronary arteriosclerosis with degeneration of the posterior wall of the left ventricle, as well as disease of the auricular muscle."

Electric shock therapy was begun, and after the third treatment great improvement was evident in the subsidence of anxiety, disappearance of self-accusatory ideas, cheerful participation in hospital activities and practically complete absence of the former constellation of mental symptoms. The response to therapy was so satisfactory after the fifth treatment that further shocks were considered unnecessary and the patient was discharged as recovered on the twenty-fourth day of hospitalization. The electrocardiogram taken after termination of shock treatment showed no change. Since her discharge from the hospital the patient has gained wight and maintained her state of recovery.

Considerable controversy has arisen regarding the employment of convulsive shock treatment in the presence of pulmonary disease, especially with respect to tuberculosis. It is common psychiatric knowledge that tuberculosis is frequently present in patients with schizophrenia. The coexistence of mental disturbance and pulmonary tuberculosis tends to create a vicious circle, in which there is enhancement of the main difficulties encountered in each disorder. Electric shock therapy can, and does, act in many cases as a means of breaking this vicious circle. Pacella [11] cited the cases of 4 patients with latent and active tuberculosis treated with electric shock in whom there were no complications and

8. Moore, M. T.; Winkelman, N. W., and Solis-Cohen, L.: Asymptomatic Vertebral Fractures in Epilepsy: Comparison with Vertebral Fractures Due to Metrazol-Induced Convulsions, J. Nerv. & Ment. Dis. **94**:309-323, 1941.

no reactivation of the pulmonary process. Smith [9] administered electric shock therapy to an agitated middle-aged woman who had active tuberculous lesions in both lungs of several years' duration. After she had received twenty convulsive shocks, both her psychiatric and her pulmonary status improved.

In this series, 30 patients had, at the time of treatment or in their past history, some form of pulmonary disease which previously would have prejudiced the consideration of electric shock treatment, as shown in the tabulation.

| Pulmonary Disorders | No. of Patients |
|---|---|
| Chronic tuberculosis | 8 |
| Pulmonary fibrosis | 7 |
| Tuberculosis (active) | 2 |
| Tuberculosis in childhood | 2 |
| Pulmonary emphysema | 7 |
| Pleural thickening | 2 |
| Pleuritis | 1 |
| History of pulmonary abscess (11 years before) | 1 |

CASE 7.—S. P., a married woman aged 58, was first examined on May 10, 1945 and admitted to the hospital on May 24. During April 1945 she began to lose interest in her personal appearance and in her home. There developed inertia, fear of infecting members of her family as the result of pulmonary tuberculosis she had had thirty years ago, agitation, insomnia and numerous somatic complaints. For two or three weeks before admission her condition worsened, with ideas of inadequacy, hopelessness and suicide. After psychiatric examination the diagnosis of involutional psychosis was made. Physical examination showed pinpoint pupils (she was using eye drops for glaucoma); expressionless, greasy facies, with the habitus of parkinsonism; an impaired pulmonary note on percussion and moist, subcrepitant, crepitant, crackling and musical rales on auscultation; moderate sclerosis of the peripheral arteries, and a blood pressure of 154 to 180 mm. of mercury systolic and 80 mm. diastolic. The electrocardiographic report (May 26) follows: "The cardiac rate is 116 to 120 per minute with normal sinus rhythm; the PR interval is 0.14 second; the P waves are tall and prominent in leads II and III; the QRS complexes are normal; the T waves are flat in lead I and elevated in leads II, III and IV. The electrocardiographic diagnosis is tachycardia." A roentgenogram of the chest showed diffuse peribronchial fibrosis without evidence of active tuberculosis (fig. 2). Electric shock treatment was instituted, and after the third treatment notable improvement was evident. Treatment was terminated after the sixth electric shock. She had recovered completely from her depression and was cheerful, outgoing and almost vivacious. Physical examination of the chest showed lessening of the rales, and roentgenograms revealed no change. Follow-up studies have shown some improvement in the pulmonary status, and she has continued to remain well mentally.

CASE 8.—S. S., a married woman aged 37, was first examined on Jan. 11, 1946. Six weeks previously there developed an agitated depression with ideas of suicide, superimposed on a chronic anxiety-tension-phobic state. She had had tuberculosis from the age of 15 and was receiving treatment for an active lesion with open cavity in the upper lobe of the left lung (fig. 3*A*). She was hospitalized but was so agitated that she was forced to leave. It was feared her physical activity

9. Smith, L. J., cited by Pacella.[11]

would seriously affect the pulmonary lesion, and after careful study electric shock therapy was advised. The electrocardiogram was normal. From Jan. 18 to May 18, 1946 she received nineteen full convulsive electric shock treatments. After

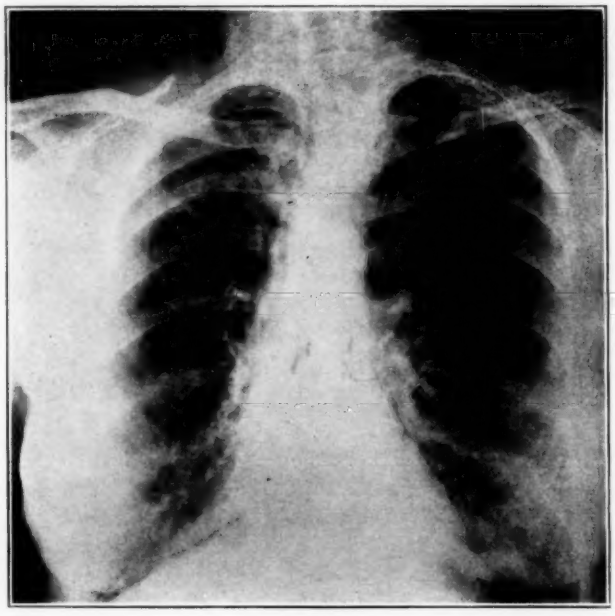

Fig. 2 (case 7).—Diffuse peribronchial fibrosis in a patient who had had tuberculosis thirty years before.

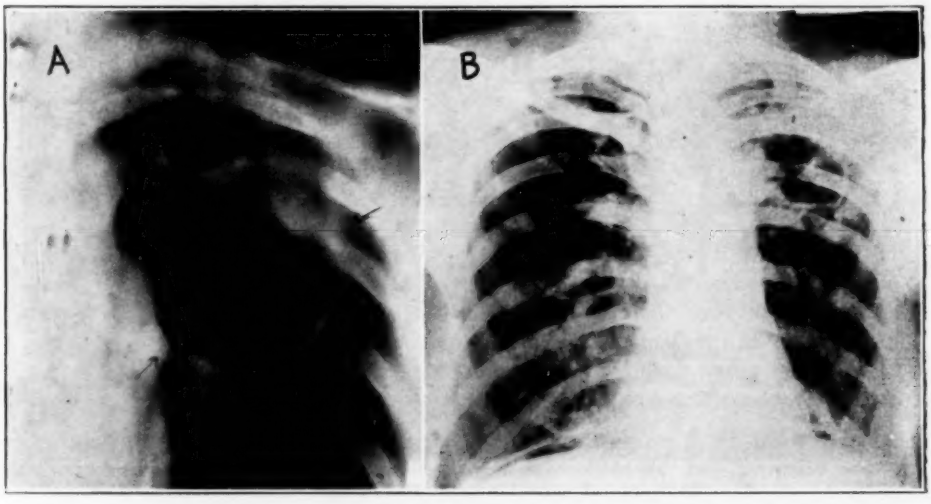


Fig. 3 (case 8).—*A*, large tuberculous cavity in the upper lobe of the left lung (Sept. 17, 1945); *B*, roentgenogram of the chest taken May 5, 1946.

her fourth treatment the agitation and depression subsided, permitting attendance for treatment at a tuberculosis clinic. A roentgenogram of the chest on May 3,

1945 (fig. 3 *B*) showed some improvement as compared with the roentgenographic appearance prior to shock therapy.

In the following tabulation are indicated a number of miscellaneous conditions found in 10 patients undergoing treatment without complications.

| Miscellaneous Conditions | No. of Patients |
|---|---|
| Peptic ulcer | 2 |
| Inguinal hernia | 2 |
| Paralysis agitans | 2 |
| Tracheotomy (tube in place) | 1 |
| Nephrolithiasis | 1 |
| Bell's palsy | 1 |
| Pregnancy | 1 |

Active peptic ulcer with a recent history of bleeding or roentgenographic signs of a crater should be sufficient reason to interdict electroconvulsive therapy. Ziegler [10] reported the case of a patient with gastric ulcer which bled after convulsive treatment. It is advisable, therefore, in cases with a history of ulcer to have proper studies made of the stomach and duodenum if feasible, to rule out active peptic ulcer before resorting to electric convulsive treatment. Inguinal hernia should not constitute a contraindication, since any form of hernia can be adequately restrained during treatment. The patient with tracheotomy was successfully treated for a depression. The pregnant patient in this series was treated when three months pregnant and went on to term, delivering a normal infant. Polatin and Hoch [11] and Goldstein and associates [12] reported the use of convulsive shock therapy during pregnancy without affecting the normal course of events.

Deaths from electric convulsive therapy may be expected, since the exact reason for such deaths is still being debated. It is to be hoped, however, that the incidence of such disasters will be materially lowered by the judicious selection of patients who require treatment and yet present attending complicating disorders. The reports in the literature thus far give electric shock therapy a cleaner bill of health than either insulin or metrazol shock treatment.[1a] Despite precautions, an occasional case will appear which will defy adequate explanation of absence of deleterious effects in a seriously complicated case, on the one hand, or of fatality in a seemingly favorable case, on the other, as illustrated in the following report.

Case 9.—The second death in this group was that of S. M., a 44 year old, physically normal man, who had been treated for manic-depressive psychosis from

10. Ziegler, L. H., in discussion on Evans, V. L.: Physical Risks in Convulsive Shock Therapy, Arch. Neurol. & Psychiat. **48**:1017-1020 (Dec.) 1942.

11. Polatin, P., and Hoch, P.: Electroshock Therapy in Pregnant Mental Patients, New York State J. Med. **45**:1562-1563, 1945.

12. Goldstein, H. H.; Weinberg, J., and Sankstone, M. J.: Shock Therapy in Psychosis Complicating Pregnancy: Case Report, Am. J. Psychiat. **98**:201-202, 1941.

Jan. 18 to Feb. 20, 1944, receiving twelve electric shock treatments. He was discharged as recovered and returned to work in an airplane factory. During October 1944 he was struck on the head by a fellow-worker and immediately thereafter became depressed, confused, agitated and hallucinated. He was admitted to the hospital on October 26. No evidence of gross organic disturbances could be found. On October 27 he was given three subconvulsive electric shock treatments, and that evening he appeared much calmer. The following morning he was found dead in bed. At autopsy the brain showed intense congestion of the pial veins in the frontal and parietal lobes and small, localized subarachnoid hemorrhages over the tips of both frontal lobes and over the left cerebral hemisphere. Histologic examination revealed congestion and petechial hemorrhages involving the frontal cortex and leptomeninges.

## SUMMARY AND CONCLUSIONS

In a series of 2,181 intramural and extramural patients receiving electric convulsive treatment many presented conditions hitherto considered contraindications to convulsive shock therapy, such as cardiovascular disease with and without hypertension, pulmonary tuberculosis, pulmonary fibrosis, skeletal disorders, cerebral arteriosclerosis, general arteriosclerosis, advanced age and organic disease of the central nervous system in the form of syphilitic meningoencephalitis. With the exception of 1 death, that of a patient with known severe cardiac disease occurring four weeks after a course of three treatments, there were no complications of moment, and the results from the psychiatric standpoint were more than gratifying. A second death occurred in a man aged 44 who offered no contraindications to treatment. Representative cases are cited in which the aforementioned contraindications existed and treatment was safely and successfully carried out.

It is felt that the former arbitrary and rigid contraindications to electric convulsive therapy should be relaxed so that many more persons may derive the benefits to be obtained from such treatment. However, there should be no relaxation in the exercise of proper caution in the selection of patients for treatment, the choice to be determined by the over-all physical status of the patient, the severity of the presenting psychosis, the immediacy of the need of treatment and the promise of a favorable result.

1813 Delancey Street.

## ABSTRACT OF DISCUSSION

Dr. Lothar B. Kalinowsky, New York: Dr. Moore rightly stresses the need to revise the contraindications to electric shock therapy. Every neuropsychiatrist has to take an occasional risk when the psychotic manifestations in themselves are more dangerous to a preexisting disease than electric shock therapy. I first saw this illustrated by a tuberculous patient in catatonic stupor; she was doomed because even tube feeding was hardly feasible and her weight had dropped to 68 pounds (30.8 Kg.). Convulsive shock treatment was started

in spite of a constantly elevated temperature. With the improvement of her mental condition her weight rose to over 130 pounds (59 Kg.).

It is regrettable to hear the frequent statement that patients have to be built up physically before electric shock therapy can be started. Actually, this treatment is the best way of increasing the weight of a psychotic patient. It is equally a mistake to preclude from treatment patients suffering from agitated depressions with hypertension. It is one of the most easily demonstrable facts of psychosomatic medicine that an abnormally high blood pressure falls when convulsive therapy removes the emotional strain of the psychosis.

Although many of us have treated patients with cardiac disease, Dr. Moore's rich material is unique. Electrically induced convulsions hardly ever influence electrocardiographic tracings.

In my opinion, the sudden death of the first patient several weeks after treatment cannot be attributed to the treatment. The second fatality has two interesting features: It occurred after a nonconvulsive treatment, which I always consider more dangerous than a convulsion-producing treatment, besides being therapeutically ineffective, and no preexisting disease was present to explain the death. This striking fact common to most of the fatalities reported is less surprising when one realizes that physical disease in epileptic patients is not aggravated by spontaneous convulsions.

I agree that abnormalities of the spine do not predispose to fracture. Dr. Moore uses tight restraint, which in my experience favors, rather than prevents, fracture of the long bones. I avoid any kind of tight restraint. Lately, I have been starting treatment with a low, subconvulsive stimulus in order to make the patient relax, and I follow this immediately with the convulsion-producing amount of current; a patient with relaxed musculature is less apt to have fracture.

It is true that peptic ulcer presents a danger, and I have seen an unrecognized diverticulitis lead to rupture and peritonitis during convulsive therapy.

A definite contraindication not mentioned in this paper is cerebral tumor, as evidenced by several cases of which I know. On the other hand, a neurologic condition without increased intracranial pressure has never been aggravated.

The elimination of most contraindications to electric shock therapy should not lead to its indiscriminate use. The treatment has few contraindications but many unpredictable complications, and the present discussion should not be concluded without a warning against such treatment when it is not strictly indicated.

Dr. Nathaniel W. Winkelman, Philadelphia: As the director of the hospital from which this report is presented, I have had the opportunity of seeing a great many of the patients reported on by Dr. Moore. Electric cerebral shock has proved of distinct value in the treatment of many of the psychoses, and it is practically specific for some. The contraindications to treatment with this modality must be carefully evaluated in every case. To deprive a patient with involutional depression, for example, of this therapy is frequently to sentence him—or, more commonly, her—to a life in a psychiatric institution; yet when this condition strikes the ravages of age may have already made themselves apparent. In the beginning my colleagues and I were probably overcautious in the selection of cases; now the pendulum has swung in the other direction, and treatments are being given in physicians' offices and in outpatient departments. For this reason, from time to time careful reevaluations must be made of the indications for and the contraindications to this means of therapy, which can do much good, but probably can also produce much harm.

A most interesting case was that of a woman physician, about 30 years of age, who was precipitated into a psychosis (catatonic schizophrenia) during her stay

in a tuberculosis sanatorium. Pneumothorax was being induced and she was transferred to the Philadelphia Psychiatric Hospital shortly after the onset of psychotic manifestations. She received both electric and insulin shock, with extremely satisfactory results, and is now practicing medicine.

Dr. Moore's results, I think, are excellent. His patients were a selected group. We do not take senile patients, patients with long-standing schizophrenia or deteriorated patients. Our results are probably much better than those in the average "run of the mill" cases for that reason.

Dr. Walter Z. Baro, Los Angeles: I should like to ask Dr. Moore whether he has the family sign a special release in all cases in which he is a little wary of giving the patient electric shock treatment. I have been with the Veterans Administration for the past eighteen months. My associates and I are handicapped by regulations and procedures, and we are supposed to give electric shock treatment only in cases in which the indications are clear.

I should also like to ask Dr. Moore whether his experience has been the same as ours, namely, that the patient with hypertension has a reduction in blood pressure during electric shock treatment. I remember a man who had continuous hypertension, with a pressure of 190 to 200 systolic, and when we were through with the treatment his blood pressure was normal.

Dr. A. E. Bennett, Omaha: In the experience of my colleagues and myself, the only absolute contraindications have been decompensated cardiac disease, suppurative pulmonary disease and generalized bacteremic infections. I believe there are further contraindications in many of the conditions Dr. Moore has described unless one uses the precaution of preliminary curarization to protect the patient from skeletal and visceral trauma. It is surprising how successful the treatment may be in cases of severe depression complicated by serious organic disease, and I agree that treatment should not be withheld because of such a complication, but that careful medical supervision, combined with the best psychiatric management, will lessen the hazards and improve the end result.

Dr. Foster Kennedy, New York: I should like to congratulate Dr. Moore on this paper. As you know, American soldiers, with rare exceptions, were not given the opportunity of this treatment by reason of its being forbidden in Washington. Many American soldiers in Britain had to be "bootlegged" over to Denmark Hill, the British hospital, in order to receive electric shock therapy. The objection of the medical profession at large, and that of a large section of the psychiatric profession, must be overcome, and it will be when the facts are plain; but facts take a great deal of digestion if there is an emotional inhibition against them.

My first patient with severe hypertension was a man aged 65 with extreme agitation and melancholia of three years' standing, who had come from South America for treatment. When I examined him, his blood pressure was 250 systolic and 140 diastolic, and his retinas were covered with hemorrhages. I refused to give him treatment. He went to Canada for six weeks, then came back with his daughters, as badly off as he had been for three years, and he said that if I did not treat him he was going to shoot himself. I treated him, not entirely because of his threat, but because I had seen, in the twenty years before the introduction of electric shock therapy, 2 cases of a condition diagnosed as "malignant" hypertensive vascular disease occurring in the course of an agitated depression cycle in which the diagnosis was disproved by events. I believe that many patients with agitated depressions have extreme hypertenesion as a result of the same hypothalamic storm as that which causes the severe depression.

I have had to treat 2 patients with multiple sclerosis. I did so with the greatest dislike and was gratified to obtain an excellent result, with improvement in their affective symptoms. This improvement may be due to the fact that their having more liberty and feeling happier made their organic structural disability, may I say, of less moment to them. The same is true in my experience with tuberculosis.

My oldest patient was 80. I have had patients in the late seventies. I have had the good fortune not to have any deaths as yet; but I think that it is pusillanimous in a physician not to have the courage to give his patient the benefit of what he can do. The surgeon has to take risks of losing his patients. The physician must have equal courage with a new medium in order to get justified results.

I do not get a signed release; I get the patient or the patient's relative to sign a permit, as he would do for having his tonsils out. Surgeons do not operate without some sort of signed permission. It is a wise procedure, and a trivial technicality, on the part of the physician to ask the same of patients who are to receive this treatment.

Dr. W. J. Otis, New Orleans: Dr. Moore has reassured us as to what one is able and is unable to do in administering treatment. In a series of 27,000 treatments given by our New Orleans group, with and without curare, there has been no fatality. A member of the family signs the usual request form for the treatment. No matter how secure one may feel in administering the treatment, the usual ritualistic orthopedic and prophylactic measures should be adhered to. In treating patients after an attempt at suicide or with orthopedic and surgical conditions, casts and other protective measurements have been employed. We have used the method as a tranquilizer in cases of the manic phase of dementia paralytica and likewise in cases of the obsession-compulsion types, with gratifying results in both. Our experience has taught us that it should be given only in a hospital under intelligent auspices by a neuropsychiatrist trained in that type of therapy. I should like to ask Dr. Moore what his results with the obsession-compulsion psychosis have been.

Dr. Eugene Ziskind, Los Angeles: I, too, wish to thank Dr. Moore for his discussion of a practical therapeutic problem. In the past year I have seen 2 patients with acute coronary thrombosis, in each instance precipitated in a person who had a prepsychotic personality, common to the involutional psychoses, and in whom, concomitant with the coronary thrombosis, there was a picture of agitated depression, raising the question of whether or not these patients did not have involutional melancholia with paranoid state in addition. I refrained from treating either patient with electric shock, and each died after two weeks of agitated depression, which all concerned felt contributed greatly to the early death. Dr. Abraham Myerson, of Boston, reported safely treating such a patient with electrically induced convulsions. I have as yet not treated such persons. I should like to hear whether Dr. Moore has had any experience with electric shock therapy in cases of coronary thrombosis.

With respect to patients with tuberculosis with induced pneumothorax, I had my first case in 1938, that of a woman with inactive tuberculosis but with pneumothorax. Her involutional depression was of two years' standing. I refused to treat her, and five years later she was still ill. Two or three years later I had accumulated requests from specialists in tuberculosis to treat 3 other such patients. I had them all admitted to the sanatorium at the same time, after I had gathered sufficient courage. The night before treatment was to start, 1 of the patients,

whose weight had fallen to 87 pounds (39.5 Kg.), bumped into a chair and sustained a pathologic fracture of the femur. I treated the other 2 patients, and they made a recovery from their depression with impunity and in a subsequent follow-up study had no active tuberculosis or mental illness. The patient with the pathologic fracture, whom I never treated, also recovered from her depression but died eight or nine months later of the tuberculosis.

The whole question is one of medical judgment, calling for individualization in each case. The picture will be seen in its entirety only at such time as an analysis is made of the cases of treated and nontreated patients, both with complicating visceral disease.

Dr. Douglas Goldman, Cincinnati: The experience of my colleagues and myself has been much like that of Dr. Moore. We have treated patients with all the complicating disorders which he has described. There are some points which I should like to mention. Patients with tuberculosis who have a large scale encroachment on their respiratory capacity with active pneumothorax, and possibly also with thoracoplasty, represent a greater than average risk. We have recently had the experience of a test dose of curare resulting in almost immediate death, before any electric shock treatment was given. Most patients who have loud heart murmurs, particularly those with rheumatic heart disease whose cardiac condition is well compensated, can be treated with impunity; but patients who have had an episode of severe decompensation must be considered to present greater risks than should be taken in some instances. We have had experience in treating patients who had severe osteoporosis, with spontaneous pathologic fractures of the vertebrae. With adequate doses of curare these patients can be treated safely, and often it is the treatment which results in their eventual recovery from the osteoporosis, because it is possible to give them adequate diet and other care. We have not considered age a contraindication to treatment, provided the patient's physical condition was reasonably good and the psychotic condition justified the treatment.

Dr. Benjamin Boshes, Chicago: Partially apropos of this paper, and partially in answer to Dr. Kennedy's remarks, I submit the followng experience: In the fall of 1942 I brought an electric shock apparatus overseas as part of hospital equipment. The hospital where I was stationed rapidly filled with psychotic patients. The shipmasters refused to accept disturbed patients for return to the States. There were few hospital ships at that time, and any available vessel had to be used. In late February or early March of 1943, after much deliberation, I began the use of electric shock treatment, which was contrary to Army regulations. It was amazing to see how rapidly the acute schizophrenic states underwent remissions. I do not pretend that these disturbances were the so-called dementia precox, but they were often severe psychoneurotic regressions. Other acute psychotic states, including the toxic deliriums incident to acute disease and trauma, responded to this therapy.

I reported my results first in a conference in Oran, Algeria, in September 1943 and subsequently published a paper in the *Medical Bulletin of the Mediterranean Theater*, entitled "Battle Neuroses: Electroshock Treatment of Refractory Cases in the Theater of Operation." The editors apparently thought kindly of the contribution and asked that the paper be forwarded to the United States. It was, and the paper was placed on the agenda of the American Psychiatric Association for its meeting in the spring of 1944. Suddenly, ten days or so before the meeting, I received a letter through official channels stating that such a paper could not be published in the United States because the therapy was contrary to Army

regulations. I canceled the paper on the program. Subsequently, I received a note from General Menninger explaining why the paper could not be published in the States. Dr. Menninger was very kind, but he had to follow regulations.

I sent some of the men with affective disorders back to duty, just as I had sent persons in private practice back to work, and traced them for two or three years. They did well in combat. They held up as well at least as the average person. Some of the men went through as high as three hundred days of combat, without subsequent break. One man required two brief courses of electric shock treatment. The treatment was effective and useful.

Soldiers responded rapidly to electric shock therapy, requiring few treatments as compared with patients in civilian life. In almost three years of psychiatric work overseas, it was my experience that once we had started the use of electric shock we never had to feed a patient by tube or evacuate any one to a ship who required restraint or any unusual care.

DR. MATTHEW T. MOORE, Philadelphia: It is a routine procedure in our institution to have a patient, or the relative, sign permission for treatment. I think I have already answered the question about hypertension.

My results with the obsessive-compulsive psychoneurosis have been uniformly poor. Work recently released from our institution regarding the treatment of various types of psychoneuroses with electric shock has shown that patients who present anxiety-tension states respond fairly well. On the whole, however, we are veering away from the use of electric shock therapy of the psychoneuroses.

Psychiatry is just emerging from its status of a purely descriptive, and somewhat nebulous, branch of general medicine. Only recently has it been able to offer medical men and the general public tangible, pragmatic instrumentalities in the way of treatment, such as narcohypnosis and shock therapy.

The surgeon when faced with the problem of an acute, ruptured appendix, a gangrenous segment of bowel or a pulmonary lesion which requires lobectomy does not hesitate to operate because he knows there is a degree of morbidity and mortality attendant on these procedures. The surgeon is a robust person mentally, and usually physically, and he does not hesitate to drive forward when he realizes that active therapy is essential. He utilizes his skill, experience and judgment where and when indicated. The psychiatrist can do the same. Then let us get down to business and face the responsibilities incident to electric shock therapy.

# ELECTROENCEPHALOGRAPHIC STUDY OF ELECTRIC SHOCK THERAPY

## Psychotic Patients Treated in a United States Naval Hospital

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IT IS recognized that electric shock therapy produces recovery in a surprising number of mentally sick people, but not a great deal is known about the exact healing mechanisms. In a previous study [1] a broad clinicotheoretic approach was made to this problem, attempting to integrate and correlate various physiologic and psychobiologic factors in the curative process. The present paper is confined primarily to the sequence of neurophysiologic events reflected in the electroencephalographic series. Always keeping in mind that the electroencephalogram registers only the physiologic state of the brain and its disturbances, and chiefly the activity from the convexity of the cerebrum, one can still derive a great deal of information about what occurs when the patient is subjected to a series of electric convulsive treatments.

### MATERIAL AND METHODS

*Material.*—Our patients comprised a series of 106 psychotic men—Naval, Marine and Coast Guard personnel—evacuated from the Pacific area. Most of them had been hospitalized for two to six months before therapy was instituted, but none had been ill longer than one year. In general, all were manifestly psychotic, their psychoses falling into the schizophrenic, schizoaffective and depressive reaction types. The age range was from 18 to 40 years; in general deviations in prepsychotic personality were not as prominent as in civilian groups; and a history of convulsions was not elicited in any case.

*Method of Treatment.*—No rigid therapeutic regimen was followed. Each patient was given what appeared to be the optimum number of treatments for him—which varied from six to thirty, as the case might be. In general, patients with depressive reactions received about three treatments a week, until a total of eight to twelve was given. The patients with pronounced schizophrenic and profound regressive reactions were usually first given daily treatments; the fre-

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† Lieutenant (jg) (MC), U. S. N. R., on inactive duty.

1. Moriarty, J. D., and Weil, A. A.: Healing Mechanisms in the Shock-Treated Neurotic Patient, J. Nerv. & Ment. Dis. **101**:205-214 (March) 1945.

quency was then gradually decreased, but the treatments were continued until about twenty to twenty-five had been given.

Two special factors in our technic of treatment must be emphasized: (1) We used unidirectional current throughout the series (Reiter electrostimulator); (2) one shock electrode was applied to the right temple and the other to the vertex. Thus, in every case the same hemisphere (the right) received the primary stimulation. With this technic, a dose of 60 to 80 milliampere seconds (delivered by about 35 volts) is ordinarily sufficient to produce a convulsion—in contrast to the much larger doses (usually 200 to 400 milliampere seconds or more) required with the "standard" bitemporal application of alternating current.

*Results of Treatment.*—With the procedure outlined, the therapeutic results were satisfactory. Of the entire series of 106 actively psychotic patients, 77 (73 per cent) made a "social recovery," or had a satisfactory remission, and were discharged home. On the other hand, only 29 patients (27 per cent) either did not improve or failed to maintain their improvement and were transferred elsewhere for further care.

*Technic of Electroencephalographic Study.*—Except in some of the earliest cases in the series, all electroencephalograms were taken with a six channel Grass apparatus, using fine needle electrodes. Both monopolar and bipolar recordings were routinely taken from homologous frontal, parietal, occipital and temporal areas. The following procedure was followed:

1. Preshock records were taken to determine the physiologic state of the brain of the psychotic patient before treatment and to have a later basis for comparison.

2. Records were taken at various intervals during the course of treatment to establish a sequence of events. In a few cases a tracing was taken during the first hour after treatment.

3. Further electroencephalograms were obtained at various intervals after the conclusion of the course of treatments. However, when possible, most records were taken at about weekly intervals.

*Criteria for Analysis of the Electroencephalogram.*—It is important to describe in detail the basis of interpretation, since there is considerable variation in method among electroencephalographers.

1. Considerable attention was paid to the general pattern of the record—whether it was regular and coherent, or uneven and disjointed.

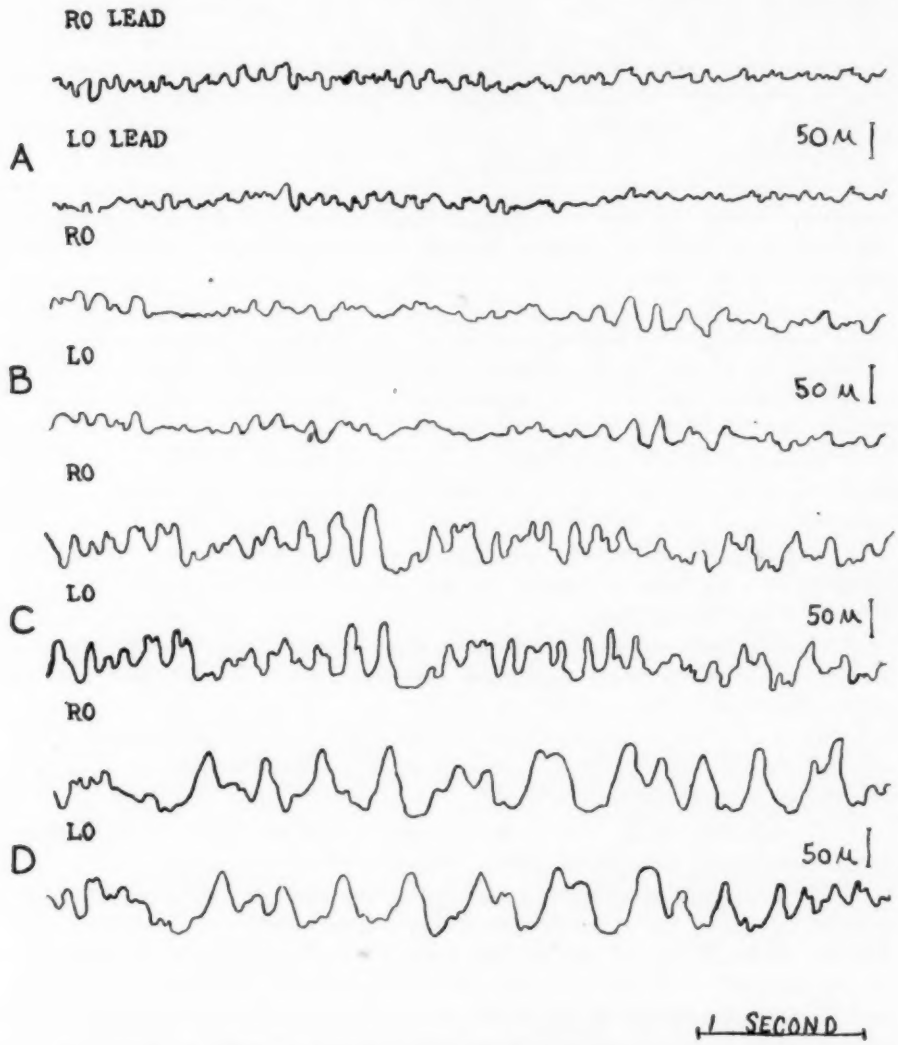
2. Most emphasis was placed on the appearance of delta activity (waves with a frequency of less than 8 per second) and on increase in voltage. However, fast activity (18 to 30 per second) of high voltage and, of course, spikes were considered definitely abnormal; but these appeared much less frequently.

3. Definite inequalities in amplitude, asynchronism of homologous areas and other signs of asymmetry were likewise interpreted as abnormal.

For the sake of convenience, the whole series of electroencephalograms was classified in four categories: normal or borderline (figure, *A*); mildly abnormal, e.g., showing some disturbance in pattern and scattered 5 to 7 per second waves of medium voltage (figure, *B*); moderately abnormal, e. g., showing considerable disturbance in pattern, with a large amount of 5 to 7 per second activity and occasional 2 to 4 per second waves (figure, *C*), and highly abnormal, e. g., showing gross fragmentation of pattern and many runs of high voltage 2 to 4 per second waves (figure, *D*).

GENERAL OBSERVATIONS

1. In the entire series of 90 patients, the preshock electroencephalograms of only 4 were definitely abnormal. This is about the same incidence of abnormality as that found in the records of normal control subjects, equated for age and for the absence of a history of convulsions.




*A*, normal; *B*, mildly abnormal; *C*, moderately abnormal, and *D*, highly abnormal electroencephalograms.

(It should be mentioned that cooperation in hyperventilation during the recording cannot always be secured from actively psychotic patients.)

2. The electroencephalogram usually showed definite abnormalities (demonstrable at least twenty-four hours later) after three to six electric shock convulsions had been administered.

A general trend toward increased abnormality with more frequent administration and increased number of electric shocks could be demonstrated; i. e., the disturbance of brain waves tended to vary directly with the number and the frequency of the treatments. However, this was true only in general, and for 1 patient no abnormalities in the electroencephalographic series were registered at any time during his recovery, with a course of fourteen treatments. Another patient showed a similarly stable pattern of brain waves during his recovery, with eight treatments. In an occasional case the record reached its maximum abnormality early during treatment and subsequently fluctuated or showed less abnormality during the remainder of the course.

3. The rates of recovery of the electroencephalographic patterns after treatment were arbitrarily classified as rapid, medium and slow. The first type included the records which returned nearly to normal in less than a week; the second, the records (the largest number) which showed considerable subsidence of abnormalities within two weeks, and the third, a few in which definite abnormalities persisted over a month (and perhaps longer).

4. Data obtained with our special shock technic reveal that, although the same hemisphere received the primary stimulation each time, the electroencephalographic disturbances which developed were entirely symmetric and equal in the two hemispheres. In other words, the electroencephalographic abnormalities appear to be due mostly, if not entirely, to the convulsion itself rather than to the electric current which produces the seizure. The one exception to this was in the area of the shock electrode. In about one third of the patients a definite asymmetry in amplitude developed in the temporal areas: i. e., the right (or "shocked") temple showed an appreciably lower voltage, or "flatter" recording, than its homologous area. This abnormality tended to disappear in the post-treatment period, together with other deviations from normal. No persistence of "shock bursts" of high voltage slow waves in the areas of application of the shock electrode was ever found, in contrast to the reports of some investigators. The absence of shock bursts in our records was probably due to the lower voltage and amperage used in our technic.

### COMPARISON OF DATA FOR RECOVERED AND FOR UNRECOVERED PATIENTS

Because of the many variables involved, it is difficult to compare the electroencephalograms of patients who recovered and those who did not. However, some noteworthy trends may be profitably mentioned.

1. The average number of treatments given the patients who recovered was almost the same as that given the patients who failed to recover, being fifteen for the former and sixteen for the latter.

2. The degrees of abnormality that developed in the electroencephalograms during the therapeutic course showed the same distribution curve for the two groups, varying with the number and the frequency of the treatments.

3. Likewise, the rate of return of the electroencephalogram to normal after cessation of therapy showed no significant variation between the recovered and the unrecovered patients.

4. The question arises whether there are prognostic implications in the electroencephalograms of psychotic patients being treated with electric shock. On the basis of a small series of cases, Proctor and Goodwin [2] found that patients with somewhat slow preshock records tended to exhibit prominent delta activity during the course of shock treatments and that none of those patients who did exhibit "3 plus delta" activity showed clinical improvement. On the other hand, Bagchi and associates [3] concluded from a study of 54 cases that patients with the greatest amount of preshock electroencephalographic abnormality profited most from electric shock treatment. Turner and associates [4] stated that whereas one fourth of their patients with normal preshock electroencephalograms recovered, none of the patients with abnormal preshock records did so. In other words, the few studies of the prognostic value of the electroencephalogram in shock therapy fail to show any agreement. These conflicting reports do not surprise us, for a careful review of our series of electroencephalograms fails to disclose any significant trend in the records of the recovered patients which distinguishes them from the records of the patients who failed to recover.

5. Another, related, study of ours deals with an attempt to determine what correlation exists between electroencephalographic abnormalities and the clinical symptoms of the psychosis. A theory of the healing mechanism of convulsive therapy in the psychoses which has been advanced from time to time is that epilepsy is "antagonistic" to schizophrenia. A slightly different statement of this idea of the curative process is that convulsive therapy changes the "schizophrenic brain" into an "epileptic brain," which then proceeds to "heal toward normal." This highly fanciful theory cannot be substantiated by either clinical or electroencephalographic observations. It has already been implied in

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2. Proctor, L. D., and Goodwin, J. E.: Comparative Electroencephalographic Observations Following Electroshock Therapy Using Raw 60 Cycle Alternating and Unidirectional Fluctuating Current, Am. J. Psychiat. **99**:525-530 (Jan.) 1943.

3. Bagchi, B. K.; Harvell, R. W., and Schmale, H. T.: The Electroencephalographic and Clinical Effects of Electrically Induced Convulsions in the Treatment of Mental Disorders, Am. J. Psychiat. **102**:49-61 (July) 1945.

4. Turner, W. J.; Louringer, L., and Huddleson, J. H.: The Correlation of Pre-Electroshock Electroencephalogram and Therapeutic Result in Schizophrenia, Am. J. Psychiat. **102**:299-301 (Nov.) 1945.

the prognostic studies previously referred to that individual psychotic manifestations, such as delusions, hallucinations and disorders of mood, disappear or persist, as the case may be, practically independently of alterations in brain waves during convulsive therapy.

An even more surprising lack of correlation is found between electroencephalographic deviations, on the one hand, and sensorial disturbances, impairment of memory and confusion, on the other. Thus, a patient who recovered without showing any electroencephalographic abnormalities in a series of five recordings exhibited simultaneously more than average confusion and patchy amnesia during his course of fourteen treatments. Several patients who recovered remained alert and sensorially clear throughout treatment, despite the development of conspicuously abnormal electroencephalograms. The general trend, of course, was a lack of demonstrable correlation rather than an inverse relationship. It should be mentioned that our therapeutic technic of a unidirectional current given by a vertex-temple system of leads produces far less intellective disturbance than the conventional technic of treatment, as has been verified repeatedly both by clinical observations and by psychometric tests. As Wilcox [5] has recently pointed out, the aim of shock therapy is properly facilitation of cerebral activity rather than destruction of brain tissue.

## SUMMARY AND CONCLUSIONS

In electroencephalographic studies on a series of 106 psychotic men at a Naval hospital given varying courses of convulsive therapy with unidirectional current, the following observations were made:

1. The preshock electroencephalograms showed no more abnormalities than are found in comparable normal control records.

2. With shock treatment almost all the patients manifested disturbances in cerebral physiology easily demonstrable in the electroencephalogram. The degree of change varied roughly with the number and the frequency of treatments, but there were many noteworthy exceptions.

3. In the majority of cases the electroencephalogram showed a considerable or a complete return to normal within two weeks after cessation of treatment. In a few cases the abnormalities were more persistent.

4. A careful comparative analysis of the electroencephalograms of the recovered patients (73 per cent) failed to reveal any significant difference from the records of the patients who did not recover. It is concluded, therefore, that the electroencephalogram does not offer a prognostic guide in electric shock therapy.

5. Wilcox, P.: Brain Facilitation Not Brain Destruction the Aim of Electroshock Therapy, Dis. Nerv. System **7**:201-204 (July) 1946.

5. Likewise, no significant correlation was found between the disappearance or persistence of individual psychotic symptoms and the electroencephalographic pattern, or between the degree of sensorial disturbance and memory impairment and the degree of abnormality in the brain waves. It is thus concluded that the obvious changes in cerebral physiology registered in the electroencephalogram do not give a direct clue to the mechanism of healing in electric shock therapy.

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# ELECTROENCEPHALOGRAPHIC STUDIES FOLLOWING ELECTRIC SHOCK THERAPY

## Observations on Fifty-One Patients Treated with Unidirectional Current

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ELECTROENCEPHALOGRAPHIC studies following electric shock treatment have been described by several authors (Levy, Serota and Grinker [1]; Hughes, Wigton and Jardon [2]; Pacella and Barrera [3]; Fleming, Golla and Walter,[4] and Bagchi, Howell and Schmale [5]). It is fairly well agreed by all these authors that electroencephalographic tracings after electric shock treatment show a definite resemblance to epileptiform wave patterns (Kalinowsky and Hoch [6]). All these brain waves tracings were obtained after administration of alternating current. The only previous observation on unidirectional, fluctuating current as administered by

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1. Levy, N. A.; Serota, H. M., and Grinker, R. R.: Electroencephalographic and Clinical Studies Following Convulsive Shock Therapy of Affective Disorders, Arch. Neurol. & Psychiat. **46**:542 (Jan.) 1941.

2. Hughes, J.; Wigton, R., and Jardon, F.: Electroencephalographic Studies on Patients Receiving Electric Shock Treatment, Arch. Neurol. & Psychiat. **46**: 748 (Oct.) 1941.

3. (*a*) Pacella, B. L., and Barrera, S. E.: Some Considerations of the Electroencephalogram in the "Convulsive State" (Electrically Induced Seizures), J. Nerv. & Ment. Dis. **96**:125 (Aug.) 1942; (*b*) Sequelae and Complications of Convulsive Shock Therapy, Arch. Neurol. & Psychiat. **53**:82 (Jan.) 1945. (*c*) Pacella, B. L.; Barrera, S. E., and Kalinowsky, L.: Variations in Electroencephalogram Associated with Electric Shock Therapy of Patients with Mental Disorders, ibid. **47**:367 (March) 1942.

4. Fleming, G. W. T.; Golla, F. L., and Walter, W. G.: Electric-Convulsion Therapy of Schizophrenia, Lancet **2**:1353 (Dec. 30) 1939.

5. Bagchi, B. K.; Howell, R. W., and Schmale, H. T.: The Electroencephalographic and Clinical Effects of Electrically Induced Convulsions in the Treatment of Mental Disorders, Am. J. Psychiat. **102**:49 (July) 1945.

6. Kalinowsky, L. B., and Hoch, P. H.: Shock Treatments and Other Somatic Procedures in Psychiatry, New York, Grune & Stratton, Inc., 1946.

the Reiter apparatus (Friedman and Wilcox [7]; Friedman [8]) was reported by Proctor and Goodwin.[9] They found a significant increase in the occurrence of cortical slow wave formations in the subjects receiving alternating current as compared with the subjects receving unidirectional, fluctuating curent.

We have tried to elaborate on their observations in this study, utilizing only unidirectional, fluctuating current. Since most of the papers just mentioned did not stress the importance of a standardized time interval between treatment and electroencephalographic recording, we endeavored to make all our electroencephalographic studies at a constant time interval. Also, in order to obtain more uniform conditions, only female patients were selected. We tried to avoid, at least in the preshock electroencephalogram, obtaining our records at the time of menstruation.

## PROCEDURES

Fifty-one patients admitted to the New Hampshire State Hospital for various neuropsychiatric conditions (table 1) were asked to submit themselves to electro-

TABLE 1.—*Distribution of Neuropsychiatric Disturbances in the Series of Fifty-One Patients*

| Diagnosis | No. of Patients |
|---|---|
| Schizophrenia | 20 |
| Involutional psychosis | 13 |
| Psychosis due to alcohol | 1 |
| Manic-depressive psychosis | 5 |
| Psychosis with mental deficiency | 1 |
| Psychosis with psychopathic personality | 1 |
| Psychoneurosis | 10 |

encephalographic study the day before electric shock treatment was started and again at definitely controlled intervals after treatments ceased, namely, four days after treatment and, again, ten days later.

Electric shock treatments were given three times a week. The number of treatments varied from three to twenty. We believe that with schizophrenic patients administration of more than ten treatments is usually advisable (Kalinnowsky [10]); however, for various reasons, not all patients in this series received that many treatments.

7. Friedman, E., and Wilcox, P.: Electrostimulated Convulsive Doses in Intact Humans by Means of Unidirectional Currents, J. Nerv. & Ment. Dis. **96**: 56 (July) 1942.

8. Friedman, E.: Unidirectional Electrostimulated Convulsive Therapy, Am. J. Psychiat. **99**:218 (Sept.) 1942.

9. Proctor, L. D., and Goodwin, J. E.: Comparative Electroencephalographic Observations Following Electro-Shock Therapy, Am. J. Psychiat. **99**:525 (June) 1943; Clinical and Electro-Physiological Observations Following Electroshock, ibid. **101**:797 (May) 1945.

10. Kalinowsky, L.: Electric Convulsive Therapy with Emphasis on Importance of Adequate Treatment, Arch. Neurol. & Psychiat. **50**:652 (Dec.) 1943.

For the purpose of this study, the electroencephalograms were rated as normal, questionably normal, pathologic and grossly pathologic. The following criteria were used in making these ratings:

*Normal.*—Normal records; records slightly less stable, or records showing atypical ocular movements.

*Questionable.*—(1) Occipital alpha index less than 10 per cent; (2) some discrepancies between the two hemispheres; (3) extremely "choppy" records; (4) single slow wave activity, between 6 and 7½ per second, without increased voltage, or (5) high frequency runs of less than 25 microvolts.

*Pathologic.*—(1) Single slow wave activity, between 6 and 7½ per second, with increased voltage; (2) frequent single slow waves, with a rate of less than 6 per second; (3) runs of 6 or 7½ per second waves, or (4) traumatic spikes of less than 30 microvolts; (5) rapid breakdown of pattern during hyperventilation, or (6) high frequency runs of more than 25 microvolts.

*Grossly Pathologic.*—(1) Spike and wave formations; (2) paroxyms and runs of waves slower than 6 per second; (3) traumatic spikes of high voltage, or (4) high frequency paroxyms of more than 50 microvolts.

We used a three channel Rahm electroencephalograph, writing on thermal Contax paper. Electrodes were applied to the frontal, motor and occipital areas bilaterally, with the indifferent electrode from the ear lobes. Hyperventilation was conducted for two minutes. All tracings were obtained with the patient lying down.

The results of our electroencephalographic studies are summarized in table 2.

## OBSERVATIONS

The preshock electroencephalogram was normal for 29 patients (59 per cent), questionable for 14 patients (27 per cent), pathologic for 4 patients ( 7 per cent) and grossly pathologic for 4 patients (7 per cent). The highest percentage of questionable, pathologic and grossly pathologic per shock records was obtained from our manic-depressive patients (80 per cent), closely followed by 65 per cent from our schizophrenic patients; only 38 per cent of our patients with involutional psychosis and 20 per cent of our psychoneurotic patients showed questionable, pathologic or grossly pathologic preshock records. The result for the last group is not quite in keeping with the observations of Pacella, Polatin and Nagler,[11] who reported abnormal electroencephalographic patterns in 64 per cent of patients with obsessive-compulsive neuroses.

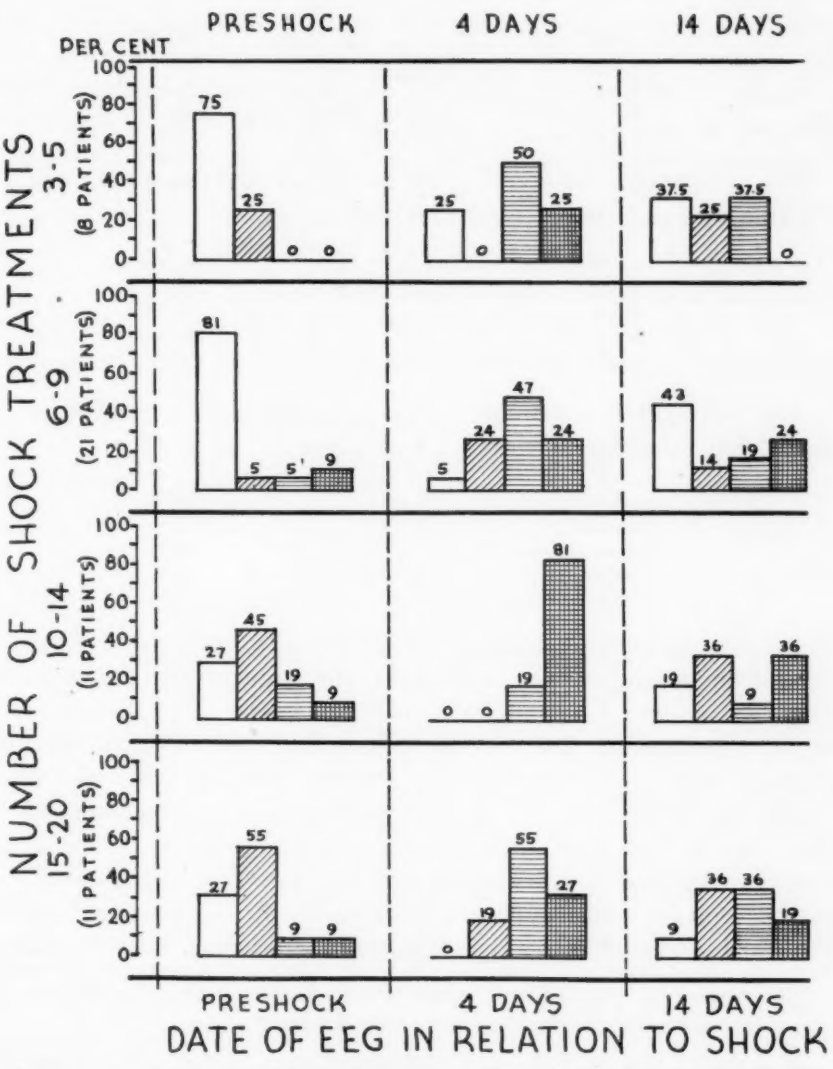
After treatment an increase in voltage, a decrease in frequency and the appearance of various abnormalities were almost universal. In general, the increase in abnormality of the record was directly proportional to the number of treatments (figure). The percentage of pathologic and grossly pathologic records increased sharply when more treatments were administered. This relation was especially noticeable in the electroencephalogram made four days after shock treatment, whereas that made

11. Pacella, B. L.; Polatin, P., and Nagler, S. H.: Clinical and EEG Studies in Obsessive-Compulsive States, Am. J. Psychiat. **100**:830 (May) 1944.

TABLE 2.—*Results of Electroencephalographic Studies Following Electric Shock Therapy*

| Patient No. | Age, Yr. | Diagnosis | Preshock Electroencephalographic Grading | No. of Electric Convulsive Treatments | Post-Treatment Data | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | Four Days | | Fourteen Days | |
| | | | | | Electroencephalographic Grading | Clinical Condition | Electroencephalographic Grading | Clinical Condition |
| 1 | 37 | Psychoneurosis, neurasthenia | Normal | 3 | Normal | Improved | Normal | Improved |
| 2 | 29 | Schizophernia, catatonic | Questionable | 4 | Pathologic | Recovered | Questionable | Recovered |
| 3 | 61 | Psychoneurosis, reactive depressive | Normal | 4 | Pathologic | Much improved | Pathologic | Much improved |
| 4 | 26 | Psychosis with psychopathic personality | Normal | 4 | Normal | Improved | Normal | Improved |
| 5 | 50 | Involutional psychosis, paranoic | Normal | 4 | Grossly pathologic | Improved | Normal | Improved |
| 6 | 29 | Schizophrenia, paranoid | Normal | 5 | Pathologic | Much improved | Questionable | Much improved |
| 7 | 39 | Psychoneurosis, mixed | Normal | 5 | Pathologic | Recovered | Pathologic | Recovered |
| 8 | 21 | Psychoneurosis, mixed | Questionable | 5 | Grossly pathologic | Recovered | Pathologic | Recovered |
| 9 | 45 | Involutional psychosis, paranoid | Normal | 6 | Pathologic | Much improved | Questionable | Much improved |
| 10 | 42 | Psychoneurosis, mixed | Grossly pathologic | 6 | Questionable | Recovered | Questionable | Recovered |
| 11 | 59 | Involutional melancholia | Pathologic | 6 | Grossly pathologic | Much improved | Normal | Recovered |
| 12 | 24 | Schizophrenia, paranoid-catatonic | Normal | 6 | Questionable | Unimproved | Grossly pathologic | Unimproved |
| 13 | 59 | Manic-depressive psychosis, depressive | Normal | 6 | Questionable | Improved | Normal | Improved |
| 14 | 29 | Schizophrenia, catatonic | Normal | 6 | Grossly pathologic | Recovered | Pathologic | Recovered |
| 15 | 47 | Involutional melancholia | Normal | 6 | Grossly pathologic | Recovered | Pathologic | Recovered |
| 16 | 23 | Psychoneurosis, reactive depressive | Normal | 6 | Pathologic | Recovered | Normal | Recovered |
| 17 | 38 | Alcoholism, paranoid | Normal | 7 | Pathologic | Much improved | Normal | Recovered |
| 18 | 34 | Schizophrenia, paranoid | Normal | 7 | Questionable | Much improved | Normal | Much improved |
| 19 | 36 | Schizophrenia, catatonic | Normal | 7 | Pathologic | Much improved | Normal | Recovered |
| 20 | 22 | Schizophrenia, hebephrenic | Normal | 7 | Pathologic | Improved | Grossly pathologic | Improved |
| 21 | 50 | Manic-depressive, depressive | Grossly pathologic | 7 | Pathologic | Improved | Pathologic | Improved |
| 22 | 59 | Psychoneurosis, mixed | Normal | 7 | Grossly pathologic | Improved | Normal | Improved |
| 23 | 43 | Manic-depressive, manic | Questionable | 8 | Questionable | Recovered | Questionable | Recovered |
| 24 | 57 | Psychoneurosis, mixed | Normal | 8 | Grossly pathologic | Improved | Grossly pathologic | Improved |
| 25 | 50 | Involutional melancholia | Normal | 8 | Pathologic | Unimproved | Grossly pathologic | Unimproved |
| 26 | 28 | Psychoneurosis, mixed | Normal | 8 | Pathologic | Much improved | Pathologic | Much improved |
| 27 | 43 | Involutional melancholia | Normal | 8 | Pathologic | Improved | Normal | Improved |
| 28 | 59 | Involutional melancholia | Normal | 8 | Pathologic | Recovered | Grossly pathologic | Recovered |
| 29 | 57 | Involutional melancholia | Normal | 8 | Normal | Much improved | Normal | Improved |
| 30 | 18 | Schizophrenia, mixed | Pathologic | 10 | Grossly pathologic | Slightly improved | Questionable | Slightly improved |
| 31 | 40 | Manic-depressive, depressive | Normal | 10 | Pathologic | Unimproved | Normal | Unimproved |
| 32 | 55 | Involutional melancholia | Grossly pathologic | 10 | Grossly pathologic | Improved | Pathologic | Much improved |
| 33 | 31 | Schizophrenia, mixed | Questionable | 10 | Grossly pathologic | Recovered | Questionable | Recovered |
| 34 | 54 | Manic-depressive, depressive | Questionable | 10 | Grossly pathologic | Much improved | Questionable | Much improved |
| 35 | 29 | Schizophrenia, paranoid | Normal | 10 | Grossly pathologic | Recovered | Normal | Recovered |
| 36 | 25 | Schizophrenia, hebephrenic | Questionable | 12 | Pathologic | Improved | Grossly pathologic | Improved |
| 37 | 45 | Psychoneurosis, mixed | Normal | 12 | Grossly pathologic | Unimproved | Grossly pathologic | Unimproved |
| 38 | 23 | Schizophrenia, mixed | Questionable | 13 | Grossly pathologic | Unimproved | Grossly pathologic | Unimproved |
| 39 | 20 | Psychosis with mental deficiency | Questionable | 14 | Grossly pathologic | Much improved | Questionable | Much improved |
| 40 | 22 | Schizophrenia, catatonic | Pathologic | 14 | Grossly pathologic | Improved | Grossly pathologic | Improved |
| 41 | 54 | Involutional melancholia | Questionable | 15 | Pathologic | Unimproved | Grossly pathologic | Unimproved |
| 42 | 58 | Involutional melancholia | Pathologic | 15 | Pathologic | Improved | Pathologic | Improved |
| 43 | 20 | Schizophrenia, catatonic | Grossly pathologic | 15 | Pathologic | Unimproved | Normal | Unimproved |
| 44 | 51 | Involutional melancholia | Normal | 15 | Questionable | Much improved | Questionable | Much improved |
| 45 | 30 | Schizophrenia, catatonic | Questionable | 15 | Pathologic | Much improved | Pathologic | Much improved |
| 46 | 35 | Schizophrenia, hebephrenic | Questionable | 15 | Pathologic | Improved | Pathologic | Improved |
| 47 | 34 | Schizophrenia, catatonic | Normal | 15 | Pathologic | Unimproved | Questionable | Unimproved |
| 48 | 36 | Schizophrenia, peranoid | Questionable | 16 | Grossly pathologic | Unimproved | Questionable | Unimproved |
| 49 | 61 | Involutional melancholia | Normal | 16 | Grossly pathologic | Unimproved | Grossly pathologic | Unimproved |
| 50 | 29 | Schizophrenia, catatonic | Questionable | 17 | Questionable | Unimproved | Questionable | Unimproved |
| 51 | 35 | Schizophrenia, catatonic | Questionable | 20 | Grossly pathologic | Unimproved | Pathologic | Unimproved |

fourteen days after shock returned to a more nearly normal pattern. We found, for instance, that of the patient receiving ten treatments or more not one had a normal record four days after shock, whereas in the group receiving less than ten treatments some retained the normal rating.




Relation of electroencephalographic abnormalities to number of treatments. Unshaded areas indicate normal; diagonal-lined areas, questionable; horizontal-lined areas, pathologic, and squared areas, grossly pathologic electroencephalographic tracings.

Similarly, there was a sharp rise in the number of grossly pathologic records as soon as ten or more treatments were administered. It was interesting to see how rapidly the electroencephalogram improved. For

instance, four days after shock treatment, only 6 per cent of the patients showed a normal electroencephalogram and 12 per cent showed questionable, 45 per cent pathologic and 37 per cent grossly pathologic records, whereas fourteen days after shock the percentage of abnormalities in the electroencephalograms had decreased as follows:

| Rating | Percentage |
|---|---|
| Normal | 29 |
| Questionable | 16 |
| Pathologic | 24 |
| Grossly pathologic | 21 |

The 29 patients with less than ten shock treatments showed the following ratings fourteen days after shock:

| Rating | Percentage |
|---|---|
| Normal | 41 |
| Questionable | 17 |
| Pathologic | 24 |
| Grossly pathologic | 17 |

The corresponding ratings for the 22 patients who received ten or more electric shock treatments were as follows:

| Rating | Percentage |
|---|---|
| Normal | 14 |
| Questionable | 36 |
| Pathologic | 23 |
| Grossly pathologic | 27 |

The last figures cited correspond fairly well to those reported by the users of alternating current. However, the patients with less than ten treatments showed a definitely smaller percentage of electroencephalographic abnormalities than did the patients reported on previously after treatment with alternating current. Proctor and Goodwin [9] also found a significant decrease of slow wave formation when unidirectional, fluctuating current was used instead of alternating current. Pacella and Barerra [3b] reported in a brief note that they saw only slight changes in electroencephalographic tracings obtained with the unidirectional current from patients who had had a large number of treatments. They concluded:

> This observation is in distinct contrast to the effect on the brain potentials with the use of the present standard electric shock machines.

In only 11 per cent of the records did we observe spike-dome formation, whereas in the series of Bagchi and co-workers [5] this change was observed in 40.7 per cent after the administration of alternating current. Of our electroencephalograms four days after shock, we noticed 26 per cent with long runs of slow, 3 to 7 per second, waves, in contrast to 45.2 per cent in Bagchi's [5] studies.

Our figures indicate that the abnormalities in the brain waves increase after ten treatments and probably produce longer-lasting disturbances of the cerebral rhythm.

For analysis of the effect of age, we divided our patients into three age groups, namely, from 15 to 24, 25 to 44 and 45 to 62 years. The electroencephalogram fourteen days after shock was taken as a criterion. We noted that the records of the patients of the youngest and the oldest age groups showed the greatest abnormality (table 3), whereas the patients of the 25 to 44 age group were able to retain more normal brain wave patterns after treatment. The factor of age has, in our opinion, often been overlooked by other authors. Hughes and associates [2] stated that the younger patients showed more abnormal waves but that the older patients were likely to manifest the more pronounced loss of memory.

### IMPROVEMENT IN THE ELECTROENCEPHALOGRAM AFTER ELECTRIC SHOCK THERAPY

Levy and associates [1] noticed improvement in the electroencephalogram after a course of electric shock treatments as compared with the pretreatment electroencephalogram. They were inclined to explain this

TABLE 3.—*Electroencephalograms Fourteen Days After Treatment, Correlated with Age*

| Age | Normal, % | Questionable, % | Pathologic, % | Grossly Pathologic, % |
|---|---|---|---|---|
| 15 to 24 | 20 | 20 | 10 | 50 |
| 25 to 44 | 33 | 38 | 25 | 4 |
| 45 to 62 | 28 | 11 | 28 | 34 |

as due to a decrease in anxiety and tension after electric shock therapy. We made this observation on 4 of our patients whose preshock records had been graded as "grossly pathologic." All these patients had a less abnormal record fourteen days after treatment than they had before treatment. Clinically, 1 of these patients was considered recovered; the condition of the second was improved; that of the third was much improved, and that of the last was unchanged.

For the 5 patients who had pathologic preshock records a similar phenomenon was observed; all but 1 showed a less abnormal record fourteen days after treatment. Two of these patients showed no improvement clinically; 2 were improved, and 1 was considered recovered. In view of these varying clinical results, we cannot accept the explanation given by Levy and associates and must admit that the phenomenon is open to speculation.

### ELECTROENCEPHALOGRAPHIC CHANGES AFTER HYPERVENTILATION

Although patterns indistinguishable from those seen in epilepsy occurred frequently in the postshock records of our series, the records showed less change after hyperventilation than one usually sees in the case

of epileptic patients. It is a well established fact that a large proportion of epileptic patients show a significant breakdown of pattern after hyperventilation. Of our electroencephalograms taken four days after shock, only 6 showed this breakdown. This is less than 13 per cent of our 48 patients whose records were classified as other than normal four days after the last treatment. Of the electroencephalograms made fourteen days after shock therapy, only 2 retained a significant breakdown during hyperventilation, or less than 6 per cent of all patients who showed records other than normal fourteen days after the last treatment. We feel that this fact serves as a distinguishing feature in the differentiation of the electroencephalogram of epilepsy and that following electric shock.

### CLINICAL CORRELATION OF THE PSYCHIATRIC STATUS AND THE ELECTROENCEPHALOGRAM

Our psychiatric results were graded as follows: No improvement, when there was no improvement at all or very slight improvement; improvement, when the patient still retained definite psychotic or neurotic characteristics despite some clinical improvement, and much improvement when the patient was able to make a social adjustment but retained minor psychopathic reactions and had gained little insight; the patient was classified as recovered when he had gained insight and complete social adjustment without noticeable psychiatric defects.

Proctor and Goodwin,[9] working with the same type of current as we did, reported in two previous communications that patients having complete remissions did not exhibit severe cerebral dysrhythmia after electric shock therapy. They also stated that patients having social remissions showed much less cortical slow wave activity than the group requiring continued care in a mental hospital. "Therefore, the appearance of cortical dysrhythmia following electric shock treatments has prognostic value."

Turner and associates [12] noted that one fourth of the patients with a normal pattern in the electroencephalogram taken before electric shock treatment left the hospital with their disease much improved or quiescent. "None of those with abnormal records prior to electric shock therapy were able to leave the hospital." Bagchi and associates [5] stated that there was a suggestion that patients with abnormal preshock electroencephalograms profited more as a group from shock treatment than those with normal electroencephalograms. This seems to be contradictory to the statements of Turner,[12] although both authors used alternating current.

Hughes and co-workers [2] found no direct correlation between the development of abnormal potentials and either the disappearance of psy-

12. Turner, W. J.; Lowinger, L., and Huddleson, J. H.: The Correlation of Pre-Electroshock Electroencephalogram and Therapeutic Result in Schizophrenia, Am. J. Psychiat. **102**:299 (Nov.) 1945.

chotic symptoms or the occurrence of loss of memory and confusion. In checking these somewhat contradictory reports, we made the following observations:

*Relation of Number of Electric Shocks Required to the Preshock Electroencephalogram.*—Of the 29 patients requiring less than ten treatments, only 21 per cent had preshock records other than normal. However, of the 22 patients requiring more than ten treatments, 73 per cent showed preshock electroencephalograms other than normal. This indicates that a normal brain wave prior to treatment promises a short course

TABLE 4.—*Relation Between Clinical Results of Treatment and the Electroencephalogram Fourteen Days After the Last Treatment*

| | Electroencephalographic Rating Fourteen Days After Treatment | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Normal | | Questionable | | Pathologic | | Grossly Pathologic | |
| Clinical Condition | No. | % | No. | % | No. | % | No. | % |
| Recovery | 5 | 33 | 4 | 31 | 4 | 33 | 1 | 9 |
| Much improvement | 2 | 13 | 5 | 38 | 4 | 33 | 1 | 9 |
| Improvement | 7 | 47 | 0 | .. | 3 | 25 | 3 | 27 |
| No improvement | 1 | 7 | 4 | 31 | 1 | 9 | 6 | 55 |
| Number of cases | 15 | 100 | 13 | 100 | 12 | 100 | 11 | 100 |

TABLE 5.—*Correlations Between Preshock Electroencephalogram and Psychiatric Condition After Treatment*

| | Preshock Electroencephalographic Rating | | | |
|---|---|---|---|---|
| | Normal | | Questionable, Pathologic, Grossly Pathologic | |
| Clinical Condition After Treatment | No. | % | No. | % |
| Recovery | 8 | 28 | 6 | 27 |
| Much improvement | 6 | 21 | 4 | 18 |
| Improvement | 10 | 34 | 5 | 25 |
| No improvement | 5 | 17 | 7 | 32 |

of treatment, and, conversely, an abnormal preshock electroencephalogram may indicate that a longer course of treatment will be necessary.

*Relation of Electroencephalogram Fourteen Days After Treatment to Clinical Results.*—The results shown in table 4 indicate that a grossly pathologic record fourteen days after the last treatment is usually found in patients showing little or no clinical improvement. On the other hand, patients showing normal electroencephalograms after treatment tend to show clinical improvement.

*Relations of Preshock Electroencephalogram to Clinical Results.*—Although the results are not very uniform, possibly because of the age factor (table 3), we found that patients with abnormal preshock electro-

encephalograms showed poorer clinical results than patients with normal records (table 5). It may be seen that only 17 per cent of our patients whose condition was unimproved had had a normal preshock electroencephalogram, whereas 32 per cent had had an abnormal electroencephalogram prior to treatment.

*Relation of Clinical Improvement to the Alpha Index.*—Ten of our patients, 6 of them schizophrenic, showed an alpha index of less than 5 per cent in the preshock electroencephalogram. Seven of these 10 patients showed a well marked alpha pattern in the postshock record. Tillotson and Sulzbach [13] also noticed that a low preshock alpha index rose after treatment in 25 to 50 per cent of their cases. Levy and co-workers [1] noticed that in some of their cases much beta activity prior to treatment was replaced by regular alpha activity after treatment. They thought that this might be due to alleviation of severe emotional tension. However, this change in the alpha pattern did not correspond to clinical improvement in our cases. Pacella and associates [3c] made a similar observation.

## SUMMARY

Fifty-one patients admitted to the New Hampshire State Hospital received varying numbers of electric shock treatments administered by a machine delivering unidirectional, fluctuating current. Brain waves were recorded prior to treatment, four days after the last treatment and, again, fourteen days thereafter. An analysis of the records revealed the following facts:

1. The highest percentage of abnormal records was obtained in manic-depressive patients, closely followed by that for the schizophrenic group. Patients with involutional psychoses and psychoneuroses demonstrated significantly more normal preshock records.

2. In general, an increase of abnormality in the electroencephalographic record was observed after treatment, characterized by increase in voltage, decrease in frequency and appearance of various other abnormalities.

3. The increase in abnormalities was usually directly proportional to the number of treatments.

4. The electroencephalograms of a large percentage of patients receiving less than ten shock treatments had returned to normal fourteen days after the last treatment. Of the group receiving more than ten treatments, a large proportion retained electroencephalographic abnormalities in this recording.

5. In the group receiving ten treatments or less, the electroencephalographic changes seemed to be less pronounced after shock treatment

13. Tillotson, K. J., and Sulzbach, W.: Electroshock Therapy in Depressive States, Am. J. Psychiat. **101**:455 (Jan.) 1945.

when unidirectional current was used than when alternating current was used.

6. The age groups between 15 and 24 years and between 45 and 62 years showed a much higher percentage of grossly pathologic electroencephalograms fourteen days after treatment than did the middle-aged group, between 25 and 44 years.

7. A small percentage of patients showed "improvement" in the electroencephalogram after electric shock treatments as compared with their preshock record, a fact which could not be explained and which was clinically unrelated to improvement.

8. Postshock electroencephalograms closely resembled the records of epileptic patients. However, the breakdown during hyperventilation so characteristic of the electroencephalograms of epileptic patients was rarely observed in the records of patients receiving shock treatment.

9. The following clinical relation between various changes in the brain waves and the psychiatric picture were found: (*a*) An abnormal preshock electroencephalogram usually indicated that a longer course of treatment was necessary in order to achieve clinical improvement. (*b*) A highly abnormal electroencephalographic record fourteen days after the last treatment was usually found with patients requiring further hospitalization. (*c*) A normal preshock electroencephalogram promised a better clinical result from electric shock treatment than an abnormal preshock record.

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# Special Articles

## ORIENTATION TO FORENSIC PSYCHIATRY

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WHILE many branches of the law impinge on the practice of psychiatry, only four will be considered in this paper. These are (1) wills; (2) business contracts; (3) marriage, divorce and annulment, and (4) crimes.

### WILLS

A person making a will must (1) know that he is making a will, (2) know the nature and extent of his property and (3) know the natural objects of his bounty. These are the three "tests" which, in a sense, the patient must "pass" before he is considered mentally competent to make a will. The psychiatrist must measure the subject by these three tests. Their meaning may now be considered.

1. Obviously, the testator must know what he is doing when he signs the will. If the psychiatrist is told, on a hypothetic question, that when the will was signed the patient was toxic, dehydrated, feverish, confused and unable to talk, that his hand had to be guided in signing the will, that he showed no recognition of the people around him and that he died a few hours later, the physician will probably feel that the patient did not know that he was making and signing a will. If this is so, the testator fails to pass test 1. He did not know he was making a will; hence the document is invalid.

2. The testator must know the nature and extent of his property. Not that he must know to the square inch the exact acreage of his land, nor that he must remember the serial numbers on his government bonds; but he must have a substantially accurate idea of what he owns. If the will reads, "I bequeath to my loving sister Jane my gold cuff links, to my loving nephew Claude the contents of my safe deposit box and to my loving brother Kilroy the Brooklyn Bridge," the examiner has a right to assume (unless it appears that the testator was trying to be funny) that the patient thought that he owned the Brooklyn Bridge. It would appear that he did not know the extent of his property (obviously not, if he thought the Brooklyn Bridge was part of his own property), and thus he fails to pass test 2, and the will is invalid.

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From two lectures delivered in the graduate course in neuropsychiatry, Rutgers University (N. J.), in December 1946.

3. The natural objects of a person's bounty are the people for whom he would naturally want to do favors. If he has any bounty to distribute, he would normally pass it out to his closest relatives, his warmest friends and his most loyal servants. Now, the law does not require a person to bequeath anything to these "natural objects of his bounty." He can cut them off without a penny, and the law will not presume that he lacks testamentary capacity. But it insists that he know who they are. If one is told that the testator thought that his sister Fanny was alive, when actually Fanny died thirty years ago, one is justified in assuming that he did not have a clear idea of the natural objects of his bounty. If he insists that the man who represents himself as his nephew Elmer is actually an impostor, when all the evidence indicates that Elmer is only Elmer, one can safely say that the testator did not know one of the objects of his bounty. Thus, he fails to pass test 3, and the will is invalid.

An important corollary to these criteria is this: The defects in question must arise because of disease of the mind. If the testator did not recognize Elmer because Elmer had been away from home for ten years, this would not invalidate the will, since the nonrecognition would arise from a simple extraneous circumstance, and not from disorder of the mind.

*Effect of a Psychosis.*—The fact that the testator was psychotic when he made the will does not necessarily invalidate the document. The claimant must prove that the psychosis impaired one of the three essential elements of testamentary capacity. It has been held [1] that an inmate of a psychiatric hospital may make a perfectly valid will. "It is not the medical soundness of mind that governs," explained the court in the Whitemarsh case,[1] "but rather testamentary capacity as defined by law. The fact that the testator was committed to an institution for the insane does not justify the assumption that he lacked legal mental capacity." Nor does a formal adjudication of psychosis serve to invalidate the will. The law recognizes that a patient (in a severe depression, for example) might be sick enough to require treatment, institutionalization, even the appointment of a guardian; yet might still know the nature and extent of his property and the natural objects of his bounty. As one court [2] put it, "The distinction between an adjudication of insanity and an adjudication that he is incompetent to dispose of his property is a substantial one."

On the other hand, a psychosis does invalidate a will if it touches on one of the three elements of testamentary capacity. If, for example, the testator has paranoid delusions (not simple prejudices, but delusions of paranoid intensity) against one of his relatives, the psychosis affects

1. *In re Whitemarsh's Estate (N. Y.)*, 234 N. Y. S. 505; *Livandais* v. *Bynum*, 116 So. 223.

2. *Waters* v. *Waters*, 207 N. W. 598 (Iowa).

his understanding of the natural objects of his bounty. Similarly, delusions of great wealth or poverty might cause failure under test 2. In such circumstances the will would be invalidated.

*Senility.*—The boundary between simple senility and senile dementia is a vague one; yet it is the psychiatrist's responsibility to draw the line. "Extreme age, mental sluggishness, and defective memory do not render a testator incapable of making a will if he is able to recall to mind his property and the natural objects of his bounty," said an Illinois court.[3] The witnesses will describe the testator's mental capacity by giving anecdotes of his odd behavior. The psychiatrist has to pierce the verbal fog and determine whether the described behavior is that of a psychotic person or simply that of a man in his dotage with no psychotic delusion formation or psychotic dementia. In one case[4] a will was held lawful, although the court said that the testator "was peevish, childish and made himself obnoxious by continually repeating the stories of his early life. But all these go with advancing years . . . there was nothing in the record to show that he was so lacking in capacity as to be unable to remember and identify his property or unable to remember and identify the natural objects of his bounty." Always, the psychiatrist must return to the three basic criteria and use them as his yardstick. No matter how psychotic the patient, if he passes these tests and if his delusions do not impinge on these specific qualities, the will is valid.

*Spiritualism.*—Sometimes as a man (or woman) grows old he seeks solace by trying to communicate with friends who have long since died. A belief in spiritualism itself will not invalidate a will,[5] since obviously such a belief does not necessarily affect the testator's knowledge of his relatives or of his property. On the other hand, if "the testator was laboring under a delusion that the spirits of the dead were directing him in all his business" the will would be invalid,[6] since the delusion goes beyond belief in spiritualism and affirmatively affects the actual process of making the will. (In this case, the testator obtained advice from the spirits as to how he should dispose of his property.)

*Alcoholism and Drug Addiction.*—Proof that a testator was chronically alcoholic or a drug addict will not, by itself, overturn a will. The authoritative Alexander[7] puts it this way:

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3. *Forberg* v. *Maurer,* 168 N. E. 308 (Ill.).

4. *In re Cooper's Estate,* 206 N. W. 95 (Iowa).

5. *Franzman* v. *Nalty,* 271 S. W. 1034 (Ky.); *Whipple* v. *Eddy,* 161 Ill. 114; *in re Chaffin's Will,* 32 Wis. 564; *Scott,* v. *Scott,* 212 Ill. 603; *Gass* v. *Gass,* 22 Tenn 277; *Brown* v. *Ward,* 53 Md. 376, and many others.

6. *Middleditch* v. *Williams,* 45 N. J. Eq. 726.

7. Alexander, J. E.: Commentaries on the Law of Wills, San Francisco, Bender-Moss Company, 1917-1918, vol. 1, sect. 475, cited by the court in *Payne* v. *Chance,* 4 S. W. (2d) 328 (Texas).

A person through excessive use of drugs or drink may become so obscured that he is, for the time being, comparable to a mad man. In such a condition he cannot make a valid will. But the effects of alcohol and drugs wear off, and though they may leave the user weakened in mind and body, yet so long as there has not been a destruction of the mentality which the law requires for making a will, it cannot be said that the fact that the testator is addicted to drinking or drugs incapacitates him from making a will.

As a matter of fact, a will made while the testator was drunk was admitted to probate in a case [8] in which it appeared "that his drunkenness did not prevent him from knowing what he was about." The evidence indicated that while the man was drunk he knew he was making a will, knew who his relatives were and how much property he had. On the other hand, the courts will be suspicious of any will signed while the testator was drunk, especially if an heir was with him at the time. Generally speaking, if a testator was under the influence of drugs while making his will, the document will be considered invalid [9] unless the defense can show affirmatively that the testator met the criteria for legal capacity.

*Ideas of Marital Infidelity.*—Senile psychotic persons often have delusions of marital infidelity. How about the will of an old person if ideas of infidelity are the only outspoken manifestations of the mental disturbance? The courts draw a line between a belief in the wife's unfaithfulness based on misinterpretation of external circumstances and a belief based on a psychotic delusion. The latter invalidates the will because it comes under the head of test 3. In one such case, the Court [10] said:

. . . to justify the rejection of the will, it must be established that the false belief is the figment of a deranged mind and not the result of an impression produced by extraneous circumstances. The burden is on the petitioner to prove the non-existence of the extrinsic evidence on which the belief rested.

*Whims and Prejudices.*—A person is entitled to enjoy a wide range of unreasonable prejudices, crackpot ideas and bizarre notions without sacrificing his testamentary capacity. Nephew August may attack the will which disinherited him on the grounds that Aunt Agatha cherished an unreasonable belief that August was the very devil because he had a nose shaped just like her uncle's. If the psychiatrist finds that Aunt Agatha literally thought that August was a devil, this would be a psychotic delusion touching on test 3 and might invalidate the will. But if, as is more likely, Aunt Agatha was using "devil" in a figurative sense, then her unreasonable prejudice does not upset the testament. So, an unreasonable prejudice against the wife in the McDowell case [10]

8. *Pierce* v. *Pierce*, 38 Mich. 412.
9. *Thomas* v. *Young*, 22 Fed. (2d) 588 (D. C.).
10. *In re McDowell's Will*, 140 Atl. 281 (N. J.).

did not invalidate the will. As a matter of fact, the courts accept a psychosis as an explanation only as a last resort, only if the testator's conduct "did not admit of explanation on any other ground.[11]

*Undue Influences.*—It is not the psychiatrist's responsibility to evaluate undue influence. Only an influence which destroys the testator's free agency is an undue influence. It must be, in effect, a substitution of one person's will or intention for another's. The psychiatrist may be able to help the court by indicating whether a person of the temperament described would be unusually gullible or unusually stubborn. For example, if the evidence indicated that one testator had a conversion hysteria and another had a paranoid delusion, the psychiatrist could point out that the former person was far more likely to be responsive to a friend's influence than the latter.

*The Psychiatrist's Role.*—In most medicolegal cases the physician examines the patient and testifies as to his findings. In cases of will litigation the psychiatrist rarely has the chance to examine the testator, who is necessarily quite dead when the case comes to court. The practitioner usually has to depend on a hypothetical question describing the testator's behavior and to conclude from that whether the subject could have passed the three tests on the day he signed the will. The psychiatrist confers with the attorney in advance of the trial and indicates what facts he will need for an honest opinion. The physician will want to study each of the three tests and ask the lawyer to give him facts focused squarely on these points, as well as general information about the patient's conduct. It is the lawyer's job to find witnesses who have enough knowledge of the testator's behavior to present an adequate description to the court. These facts are then assembled into the hypothesis which is given the psychiatrist, and on which he bases his conclusion as to the mental capacity of the testator.

In the rare cases in which the lawyer has the client examined before the will is prepared, the psychiatrist's job is, of course, much easier. In addition to the routine mental examination, the psychiatrist will want to analyze the patient's thinking from the point of view of the three tests. For example, he will ask the patient to name the members of his family and to indicate their relationships to him, and he will later verify these statements. He will ask the patient for a brief description of each relative, to see whether any undue emotional response is produced when the patient talks about any of them. The physician will take careful and accurate notes and will preserve them. He will prepare a report for the attorney, quote freely and verbatim from the patient's own remarks and be certain that the report contains enough facts to support the conclusion about the patient's mental capacity. He will

11. *Snell* v. *Weldon,* 243 Ill. 496.

carefully preserve a carbon copy of this report and use it later (when called as a witness) to refresh his memory.

In some cases a psychiatrist who had examined the patient in life for some other purpose is called as a witness after the patient has died. If the examination had revealed no mental derangement, and if the will had been made at about the same time, the physician's opinion is, of course, of prime value to the court. If the examination had revealed a mental disorder, the chances are that the focus of the examination was not on testamentary capacity, so that conclusions as to legal mental capacity have to be made more or less inferentially. Still, this is more direct evidence than reliance on a wholly hypothetic question. Even though he had not examined the patient specifically for testamentary capacity, the physician should be in a position to give honest and accurate answers to such questions as, "Did he have sense enough to know what he was doing when he signed a document?" or "Did his delusions extend to members of his family?"

### CONTRACTS

While the physician thinks of a contract as a formal and formidable legal document, the truth is that most contracts are never put in writing. The patient who asks the physician to give him some medicine for his headache is making a contract. So is the customer who enters a store and says, "Give me a package of cigarets." A contract is not valid (except in certain special circumstances) if by reason of mental derangement one of the parties did not know what he was doing. However, the subject's ignorance of what he was doing must be based on mental derangement, and not simply on lack of sophistication or technical knowledge. If, for example, a naive young man is persuaded to buy a business for which he is unfitted, he cannot call on a psychiatrist to help cancel the contract on the grounds that he did not understand the implications of the transaction. Such misunderstanding arose from ignorance, poor judgment or lack of training, not from mental disorder. In general, a contract made by a lunatic is voidable; and if made by a regularly adjudicated lunatic it is void.[12]

*Necessities.*—Would this doctrine affect the right of a physician to collect for medical services rendered to a psychotic patient? No, because a contract to supply "necessities" to a child or to a lunatic is valid and enforceable. Medical service, like food, is universally considered a "necessity."

*Drunkenness.*—A contract made with a drunken man cannot generally be enforced against him unless it is for a "necessity." Simple over the counter sales at standard prices are usually considered enforceable contracts. The psychiatrist will be asked whether the degree of

12. *Wadford* v. *Gillette*, 137 S. E. 314 (N. C.).

drunkenness was such as to strip the participant of any understanding of the implications of the transaction; if it was not, the contract would probably be valid. If it appears that in an expansive mood the drunken person agreed to make a huge purchase, it is probable that the physician will testify that the drunkenness adversely affected his ability to understand the implications of the contract; and since this lack of understanding rose from a mental disorder (acute intoxication) it invalidates the agreement. Only the person who was drunk can petition to have such a contract canceled. The sober participant cannot escape from the contract by pleading that the other was *non compos mentis*.

*Psychosis.*—If a person is psychotic, but if the casual observer cannot see that he is insane and enters into a contract in good faith, the courts may enforce the contract. Whether they will depends chiefly on whether the deal can be dissolved without any loss being suffered. For instance, a salesman makes a contract to sell a machine to a lunatic, not knowing that the customer is psychotic. The purchaser's guardian appeals to have the agreement voided. This will usually be done if the machine can be sent back in exchange for the money paid out. However, if a plumber spends all day fixing the pipes at the request of a lunatic, it is apparent that the contract cannot be set aside without causing a loss to the plumber. The latter cannot get back his time. In such a case the contract would probably be enforced if the plumber was acting in good faith. If a patient is obviously deranged, it will be assumed that the other participant knew it and that he did not act in good faith.

*Mental Deficiency.*—It is often easy to sell a bill of goods to a mentally defective person, and the psychiatrist may be called on to help the patient escape from his ill advised financial commitment. If a child of the corresponding mental age would not have been able to understand the implications of the contract, the courts will usually set it aside, unless (as sometimes happens) it appears that the patient's maturity and actual experience with the world make him far shrewder than a child of the same mental age. A man of 50 with a mental age of 8 who has been a self-supporting day laborer all his life is not the same as a normal 8 year old child when it comes to making business agreements. Actually, he has a moderate amount of business judgment, based on years of experience in selling his labor—something that the bright 8 year old child (whose mental age might exceed that of the laborer) would lack. Thus, the psychiatrist should not depend on the parallel with a child of the same mental age in evaluating contractual capacity. The problem is a pragmatic one, to be decided independently on the facts of each transaction. For instance, I examined a mature woman with a mental age of 7½ years who was persuaded

to buy a twenty-four volume encyclopedia. The salesman dazzled her with his assurance that the cost was only 2 cents a page and that she could stretch the instalments over a year. He never told her the actual aggregate cost, and she signed the contract because 2 cents a page seemed very cheap. As the encyclopedia contained 6,000 pages, the customer was committing herself to an aggregate payment of $120, or $2.50 a week. It was testified that with her mental age she could not perform the necessary calculation in her head. (She would have had to multiply 0.02 by 6,000 and divide the product by 52 to get the weekly payment figure of $2.50.) Her failure to see the implications of her contract was rooted in a mental disorder—namely, mental deficiency. Hence the contract was voidable. On the other hand, a farmer with a mental age of 7½ years who had been selling chickens and eggs to neighbors for years could not escape the consequences of an ill advised agreement to sell eggs at a low price on the basis of his mental deficiency because in this instance, unlike the encyclopedia case, the patient's entire background indicated that he could understand such transactions. A general rule about mental age and business capacity is not used. Each case stands on its own feet.

*Senility.*—A man in his dotage conveyed his house to a seller of oil stock. The children sought to set the contract aside on the ground of lack of contractual capacity because of senility. If the psychiatrist can find evidence of a senile psychosis or senile dementia, the contract will probably be voided. If he reports simple senility, with fragmentary memory defects, childishness, irritability and a fondness for living in the past, the contract will probably stand. The psychiatrist might find some mental trait short of psychosis which specifically impairs contractual capacity. For instance, if he found that the patient was grossly disoriented, though quite free from delusions, he might testify that a man who does not know even in what century he is living can hardly understand a business transaction. In general, the doctrines applying to senility and wills apply also to senility and contracts, though less mentality is required to make a valid will than to make an enforceable contract.

*Nonwithdrawability of Action.*—Sometimes a mentally deranged person makes an agreement, repents of it, institutes a suit to set it aside and is then influenced to withdraw the suit. As a general rule, a person having once initiated action on the grounds of his own incapacity, cannot withdraw the petition,[13] because there is too much possibility that he has been prevailed on to stop the action by some one taking advantage of his mental enfeeblement.

13. *Warker* v. *Warker,* 140 Atl. 889; also, *in re Rhodes's Estate,* 136 Atl. 408, and many others.

MARRIAGE, DIVORCE AND ANNULMENT

While there are many causes for annulling a marriage, only two of them concern the psychiatrist: (1) lack of mental capacity and (2) fraudulent concealment of prior nervous or mental disease.

*Annulment for Lack of Mental Capacity.*—"The contract to marry is extremely simple. It does not require a high degree of intelligence to comprehend it." [14] That appears to be the opinion of the courts. Thus, a man may be too defective mentally to be permitted to buy a hamburger stand, yet bright enough to be permitted to make a valid marriage contract. While the standard of mental capacity required for marriage is very low, it is still necessary that the partner be free of any mental disorder (at the time of marriage) which would render him incapable of giving his free consent or which would rob him of his ability to understand the "implications" of marriage. If it appears that the person was insane at the time of marriage, he can apply to have the marriage annulled. Similarly, with a person who married while drunk. Many states have laws which forbid ex-inmates of hospitals for mental disease to marry without clearance from the authorities of the hospital. However, violation of such a law will not nullify the marriage; it merely lays the violator open to criminal penalties.

The question which the psychiatrist must answer is this: "Did the party understand the nature of the marriage relationship and the obligations assumed under it?" [15] Generally courts are reluctant to void a marriage. The evidence must be clear, and it must apply to the petitioner's mental state at the time of the marriage. If the evidence is evenly balanced on the two sides, the courts tend to let the marriage stand. On the other hand, if it is clear that the party was psychotic or so drunk or mentally defective at the time as not to understand the obligations, nature and implications of marriage, the annulment will usually be decreed.

An interesting and important question concerns the right of the competent partner to take action under this doctrine. There is no doubt that a person who finds that he married while he was drunk or psychotic can have the marriage annulled. But can the other partner institute such action? For example, a woman marries a man who impresses her with his quiet shyness, his seriousness of purpose and his freedom from vices. Later it appears that the man is a victim of hebephrenic dementia precox. He does not want to annul the marriage. She does. Can she institute action? In most states she can-

14. Singer, H. D., and Krohn, W. O.: Insanity and Law, Philadelphia, P. Blakiston's Son & Company, 1924, p. 265.

15. *Adams* v. *Scott*, 93 Neb. 537; *Hagenson* v. *Hagenson*, 258 Ill. 197; *Dunphy*, 161 Calif. 380; *Storf* v. *Papalia*, 24 N. J. Misc. Rep. 146, and many others.

not.[16] However, the New Jersey statute [16a] (revised Statutes 2:50-1) has the following reference to a suit for annulment based on lack of mental capacity: "Where the competent party is the applicant, such applicant shall have been ignorant of the other's incapacity." Presumably this means that the sane spouse can initiate action. In a recent case,[17] an annulment was granted with the following dictum, "Where defendant lacked capacity to marry and petitioner was ignorant of that, and did not ratify marriage [18] after knowledge of that lack of capacity, he is entitled to a decree of nullity." A similar doctrine seems to prevail in Missouri, where, at the petition of a husband who had lived with his wife almost twenty years, a marriage was annulled when it was shown that she had never had sufficient mental capacity to make a marriage contract.[19] Similarly, in New Hampshire a marriage was annulled at the petition of the competent spouse when it appeared that he had been kept in ignorance of the mental status of his fianceé until after the ceremony.[20] Although the apparent ground in this case was fraud, the legal implication is certainly that it was the lack of capacity which nullified the marriage, since an insane person could hardly have engaged in wilful deception. The law on this point—the right of the competent spouse to institute suit—seems to be in a state of flux. Originally, the same rule applied to marriage as to all contracts attacked on the basis of lack of capacity, namely, that only the incompetent party could start suit. However, there appears to be a growing liberalization, the effect of which eventually may be to allow either spouse to open the question.

If one party was drunk during a marriage ceremony, the marriage is voidable at petition of the spouse who was drunk provided that the intoxication was enough to rob him (or her) of the required degree of understanding. That the participant was simply "a bit high" will not be sufficient to void the marriage.[21] At present there appears to be no way in which the sober spouse can void such a marriage at his (or her) own petition. Even if the decision in *Storf* v. *Papalia* [17] is sustained, it would not open the door to such actions, because the essential feature in that case was the husband's ignorance of his wife's mental capacity, whereas it is unlikely that a sober man would not realize that his marriage partner was drunk. If a couple marry while one is drunk but continue to live together for any time after the return

16. Thus, *Sleicher* v. *Sleicher,* 228 N. Y. S. 711, and *Hoadley* v. *Hoadley,* 155 N. E. 728.

16a. N. J. Rev. Statutes **2**:50-1.

17. *Storf* v. *Papalia,* 24 N. J. Misc. 146.

18. Ratify here means to continue to live with the spouse as man and wife.

19. *Chapline* v. *Stone,* 77 Mo. App. 523.

20. *Keyes* v. *Keyes,* 22 N. H. 553.

21. *Prine* v. *Prine,* 36 Fla. 676.

of sobriety, this is considered ratification of the marriage, and no suit for annulment on that basis may subsequently be initiated.

The psychiatrist is sometimes asked to pass on the mental state of an old man who has entered into a marriage which, his family suspects, was prompted more by the irritations of a large prostate than by the promptings of true love. The same rules apply. If a senile psychosis was present, the marriage is voidable. If the mental state was one of simple, nonpsychotic senility, the psychiatrist must determine whether the patient was "capable of understanding the nature of the marriage contract and the duties and obligations such a contract entails" (Singer and Krohn [14]).

Since a psychoneurosis does not deprive a person of mental capacity, a suit to annul a marriage on that basis cannot be initiated by either party.

One other type of lack of capacity may interest the psychiatrist. That is impotence on an emotional basis. The capacity to marry implies a capacity to perform sexual intercourse. A permanently impotent single man is incapable of contracting a valid marriage (except possibly if he had explained that incapacity to his prospective wife and she accepted him in spite of it). If a woman does not know of the existence of a permanent impotence in her fiance, she can ask to have the marriage annulled after she discovers the defect. The impotence may have an emotional basis. However, since it is a simple question of lack of capacity, not a matter of misconduct or wilfulness, the impotence invalidates the marriage regardless of its cause. In a divorce action for constructive desertion (page 742) the emotional nature of the impotence may be a material factor; but not in an action for annulment, where it is relevant only so far as it touches on the question of the permanence of the defect.

*Annulment for Fraudulent Concealment of Nervous or Mental Disease.*—Fraud implies a wilful intent to deceive. If a spouse deliberately perpetrates a fraud, if the fraud goes to the "essence" of the marriage and if the other partner relied on the misrepresentation, the marriage will usually be annulled. Misrepresenting vocational, social or financial status is not such a fraud as goes to the "essence" of a marriage. Consequently, this kind of misrepresentation is not a valid basis for annulment once the marriage has been consummated. On the other hand, wilful concealment of serious ill health is a fraud that goes to the "essence" of a marriage. Generally the concealment of prior mental disease is not considered a fraud because the mental disorder itself makes the partner incapable of guilty deception. Even if a person has recovered from a psychosis prior to marriage, silence about this is not a fraud because a person recovered from a psychosis rarely considers that he was insane. If he does not think he was ever insane,

then it is scarcely fraud if he does not say so. The result of this doctrine is to make it almost impossible to get an annulment on the grounds of fraud if concealment of mental disease is the basis of the charge.

An excellent illustration is the case of *Buechler* v. *Simon*.[22] In 1906 a young woman spent seven months as a patient at an institution for mental disease. She was discharged as "cured." In 1925 she married. In 1926 an involutional psychosis developed. Now, for the first time, the husband learned of her previous experience in a psychiatric hospital. He contended that his wife's concealment of that was a fraud. The court refused to annul the marriage, using the following informative language:

> The defendant had made no affirmative representation as to her condition. Silence resting on honest belief, even in things false, is not actionable. The defendant was not aware that her nervous breakdown was an attack of insanity. By the time she was married, 20 years later, the breakdown of 1906 was an unpleasant episode and the commitment an accident in the drama of life which she had long since put aside without thought or suspicion of recurrence. When she omitted to recount that experience, it was not a conscious concealment. She did not simulate health, she felt it.

The touchstone of this case can be applied in almost any action to annul on this basis. Still undecided is what would happen if a psychiatrist told a patient that she had recovered from a psychosis and that she should not marry without apprising her fiance of that fact. If the woman married and, in contravention of the physician's advice, concealed her psychiatric history, then it is possible that an annulment might be granted, because the factor of honest error (so prominent in the case of *Buechler* v. *Simon*) would be absent. On the other hand, if she was insane when she married, an action for fraud could not be maintained because she would not have mental capacity to perpetrate a wilful fraud.

Wilful concealment of a seriously disabling or potentially hereditary nervous disorder would be a fraud if the patient were sane. This is illustrated in the case of *Busch* v. *Gruber*.[23] Here, an epileptic man, under constant medical treatment, concealed the epilepsy and affirmatively assured his fianceé that he was in good health. Indeed, he boasted that he had never had occasion to visit a physician. After marriage the wife saw him in a fit, learned of the epilepsy and left him. Had she remained with him, she would have ratified the marriage and barred an action to annul. As it was, the court said,

> When a man has been suffering from epilepsy but represents that he has never been sick and when after the marriage the wife discovers the disease and straightaway leaves him, she is entitled to have the marriage annulled for fraud notwithstanding consummation.

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22 *Buechler* v. *Simon*, 146 Atl. 420 (N. J.).

23 *Busch* v. *Gruber*, 131 Atl. 101 (N. J.).

Concealment of previous commitment to a psychiatric hospital is not in itself a fraud except (*a*) when the person was sane at the time of the marriage and (*b*) when he had affirmatively stated that he was never at a psychiatric hospital. Mere silence in such a case would not be fraud, because normally the recovered patient does not believe that he was insane and would have no more reason for telling of his stay in a psychiatric hospital than for cataloguing for his fianceé his entire past medical record.

*Divorce from an Insane Spouse.*—In most states, mental disease is a bar to any divorce action. Since a psychosis is a disease, the other partner has no more basis for asking for a divorce for mental disease than for expecting pneumonia or a fractured leg to be grounds for divorce. In a few states, mental disease is a statutory basis for divorce, but over most of the country this is certaintly not true. As a matter of fact, desertion, adultery and cruelty are not grounds for divorce either if the misconduct is committed by an insane spouse.[24] As the court said in the case of *Kunz* v. *Kunz*,[24] "Since insanity is no cause for divorce, nothing which is a consequence of it can be."

*Constructive Desertion.*—The psychiatrist should know that refusal to engage in or to submit to sexual intercourse is considered "desertion" in most states. Hence, if desertion is a cause for divorce, failure to engage in sexual intercourse is also a cause for divorce. The question facing the psychiatrist is whether this is wilful, since only "wilful" desertion is grounds for divorce. If a man falls into an emotional state in which he is unable to have or to maintain an erection, his wife may (after the requisite time) sue for divorce, alleging constructive desertion. The husband could defend on the grounds that his impotence is certainly not "wilful." The wife may retort that since it is not due to "organic" disease it is a mental phenomenon, and therefore related to lack of motivation, and hence to "will." The psychiatrist is asked to give an expert opinion. Most psychiatrists would state that a neurosis is a "disease" which the patient does not acquire voluntarily. Yet, on cross examination, many experts would be jockeyed into admitting that such factors as lack of motivation and secondary gain place neurosis in a somewhat different category from a disease accidentally incurred, such as typhoid.

The situation is even more ambiguous in the case of a wife's refusal to submit to sexual intercourse because of emotional blocking. Here the courts are prone to decide that, since the wife's role could be passive, she could submit if she wanted to, and therefore her refusal is wilful.

24. As in *Tiffany* v. *Tiffany*, 84 Iowa 122; *Wray* v. *Wray*, 19 Ala. 522; *Kunz* v. *Kunz*, 213 N. W. 906; *Broadstreet* v. *Broadstreet*, 7 Mass. 474, and many other cases.

It takes exceptional eloquence on the part of the psychiatrist to persuade the court that this kind of behavior is a "sickness," not under control of the will.

*Adultery.*—In all states but South Carolina adultery is grounds for divorce. The psychiatrist comes into the picture when the misbehaving spouse alleges that he is psychotic or that he (or she) is impelled to promiscuous sexual behavior by reason of neurotic personality, alcoholism or constitutional psychopathy. Where psychosis is established, the spouse is not "responsible" for the adultery, and divorce will not be granted. Such a defense as "nymphomania" or "psychopathic personality" rarely impresses the courts, though if it is genuine the psychiatrist is certainly entitled to call the court's attention to modern scientific opinion as to the factor of "wilfulness" in these disorders. Sometimes psychiatric examination of the defendant reveals a clear hypomanic state, which, being technically a psychosis, would, or should, serve as an adequate defense.

*Cruelty.*—Psychiatrists are sometimes asked to testify that the defendant's behavior caused a "nervous breakdown," a psychoneurosis, or even a psychotic reaction, in the other spouse. The difficulty is that the defense will cite evidence to show that the complainant always had a "tendency" toward nervousness, or even, indeed, that it was complainant's own neurotic personality which caused the defendant's cruelty in the first place. There is a growing tendency to liberalize the concept of "cruelty," so that in most states an emotional trauma is considered as valid a basis for a divorce suit as is a physical injury. (Thus, cases of adultery are only one-fourth as common on the divorce court docket as they were in 1870, whereas cases of cruelty are four times as common. Men are not becoming more cruel to their wives; cruelty is merely being defined more liberally.)

## CRIMINAL RESPONSIBILITY

To escape responsibility for his crime, the deranged defendant must show that "he was laboring under such a defect of reason from disease of the mind as not to know the nature and quality of the act, or, if he did know it, that he did not know he was doing what was wrong." This is the McNaghten formula,[25] and it is the basis for determination of criminal responsibility in every state of the union and in the federal courts, both civilian and military. This formulation is sometimes considered obsolete, since it was laid down in 1843 and one likes to think that psychiatry has made progress since then. However, there seems little chance that the formula will be abandoned or rewritten in the foreseeable future. As one court[26] remarked, "the formula is now

25. *Daniel McNaghten's Case*, 8 Eng. Rep. 718, House of Lords, 1843.
26. *State* v. *Mackin*, 36 Atl. 1040.

so completely imbedded in the administration of criminal law as to be considered no longer subject to challenge."

The psychiatrist's task, then, is to match the defendant's mental status with the formula and report his opinion to the jury, which, in turn, will determine responsibility. It is frequently alleged that "the law is an ass" for supposing that a person could be legally sane and medically insane, or vice versa. However, this is not the state affirmed, and only a person unfamiliar with the subject will make this criticism. When an insane man is convicted of crime he is not thereby declared to be "legally sane." He is merely held to be legally "responsible"—that is, "answerable" for his acts. The distinction between insanity and irresponsibility is perfectly valid. As every psychiatrist knows, some psychotic patients do respond to fear of punishment or promise of reward (that is, they are "responsible"—they respond); others do not. There is thus a vast sociologic difference between these two groups of psychotic persons.

In analyzing the patient's mental state, the psychiatrist must first consider what each clause of the McNaghten formula means.

*"Defect of Reason from Disease of the Mind."*—The practitioner must first determine whether in fact there was a mental disorder which impaired reasoning. If no mental disorder at all is found, it is scarcely necessary to proceed with the rest of the formulation.

*"Know the Nature of the Act."*—This means that the patient can perceive the physical characteristics of his act. The nature of the act of pulling a trigger is that something will be hurled out of the gun barrel. Almost every defendant knows the nature of his act, since only an idiot would be unaware of such a simple physical characteristic as the fact that a sharp knife cuts skin. Few subjects will fail this test.

*"Know the Quality of the Act."*—While the word "quality" is variously defined, I have elsewhere,[27] in an analysis of decisions, pointed out that the nearest definition of "quality" is the word "harmfulness." A child who turns on the gas knowing that a vapor will escape and spread through the building certainly knows the "nature" of the act of turning the jet. But if he does not know that this would be harmful to the people in the house he does not know the "quality" of the act. Or, consider this example, "Sir Fitzjames Stephen instances the case of an idiot who cut off the head of a sleeping man, remarking that it would be great fun to see him looking for it when he woke.[28] Here, the idiot undoubtedly knew the nature of the act, since the nature

27. Davidson, H. A.: Criminal Responsibility, New Jersey Law Rev. **1**:123 (May) 1935.

28. Cited by Mercier, C.: Criminal Responsibility, New York, Physicians and Surgeons Book Company, 1926, p. 215.

of the act of decapitation is that it removes the head from the body—as he obviously knew. But he did not know the harmfulness of such an act, as indicated by his remark. In fact, this is the only clause of the McNaghten formula which would permit the idiot to escape conviction. Mercier [28] expressed the belief that the third clause ("did not know he was doing what was wrong") might exculpate him; but this is unlikely, since it is probable, as Fitzjames Steven himself put it, that "the idiot would know that people in authority would not approve of this"; thus he would know that the act was wrong. The psychiatrist frequently finds that the patient knew *what* he was doing but that he had no implication of the harmfulness of his act. If this lack of realization was due to ignorance, poor education or unsophistication, it is of no help in exculpating him under the formula. But if this lack of realization is due to mental deficiency, an involuntary toxic state or a psychosis, it comes squarely under this clause of "quality."

In some states the word "consequences" is used instead of "quality" at this point in the formula. If it means "consequences to the victim" it has practically the same meaning as "quality." If it means "consequences to the perpetrator" it has no meaning, except possibly ignorance of the law.

*"Know that the Act Is Wrong.*—This is usually the clause on which the entire defense turns. The psychiatrist should know two things about the word "wrong." First, it is a concrete, not an abstract, concept. It means, "Did he know the particular act was wrong?" and not "Did he have a philosophic concept of good and evil?" Second, it implies that he knew that society considered the act wrong, and not that he himself considered it wrong. For example, a burglar says that he sees nothing wrong in stealing from rich people, who already have more money than they can use. It cannot be argued that he should be excused because he did not know that burglary was wrong. The point is that, while in his private ethical code it may not have been wrong, he did know that the law (the community) considers burglary wrong. On the other hand, suppose a victim of paranoid schizophrenia hears God's voice commanding him to kill some one. He believes that society would applaud him for following a Divine wish; and since, by reason of his mental disorder, he does not know that the act was wrong he is irresponsible.

The psychiatrist is entitled to consider such behavior as voluntary surrender to the police, statements of repentance and sorrow and flight from the scene as evidence that the subject knew that he was doing something wrong. The physician may also call the court's attention to the fact that the subject may have known that the act was wrong without realizing how wrong it was. This is especially true with respect to idiots and imbeciles, who may think of an act as being

merely naughty (such as gleefully pushing his brother out the window) when it is actually murderous. In this type of case the defendant has two possible clauses of escape. It may be argued that he did not know the quality (harmfulness) of the act, or that he did not "realize" how wrong it was. The decision rests with the jury, not with the psychiatrist, whose function is simply to place his findings before the jury, together with his own expert opinion as to whether the defendant knew (and to what extent he knew) the nature, quality and wrongfulness of the act.

Many persons who should know better will state that the test of insanity in criminal law is whether the accused knew the difference between right and wrong. This is not only an oversimplified answer; it is a misleading one. Knowing the difference between right and wrong is a matter of ethical philosophy. There was a time when this really was the test, and an unhappy time it was. Consider, for instance, the state of affairs in 1818. A 12 year old child was arrested for stealing spoons—at that time a capital offense. The defense was that he was too young to realize the enormity of the offense. However, the prosecution called the minister, who testified that the child attended church regularly and that the pastor often preached sermons on good and evil and always told his parishioners to do no evil. On this evidence, it was held that the child was guilty because he had been taught the difference between right and wrong. The court[29] said that the test was only that the defendant should "have sufficient discernment to distinguish good from evil." If the "right and wrong" test were in use now, this would indeed be a fair criterion. It is true that in some states the phrase "know right from wrong," rather than "know that the act was wrong," is used even today. But always it is used in connection with the particular act. For instance, in military law the formula is given[30] in this way: ". . . so free from mental derangement as to be able *concerning the acts charged* . . . to distinguish right from wrong." Thus, while "distinguish right from wrong" is used, the preliminary clause (italics mine) makes it clear that it must be applied with respect to the specific act, not with respect to a general ethical philosophy. Many states have similar variants in the phrasing. In California,[31] for instance, the formula is, ". . . did not know the nature or quality of the act or that it was wrong to commit it" the phrase "to commit it" serving to delimit the general concept of "right and wrong" to the specific act, just as does the phrase "concerning the

29. *State* v. *Aaron*, 4 N. J. L. 263.

30. Manual for Courts-Martial, Document 14a, United States War Department, Office of the Judge Advocate General, Washington, D. C., 1928, para. 28a.

31. *People* v. *Gilberg*, 240 Pac. 1000.

act charged" in military law. In Colorado [31] the phrase is, "no capacity to understand the nature of the act and no ability to distinguish between right and wrong as applied thereto," the final phrase again removing the test from the realm of the general and ethical to that of the specific and pragmatic. The psychiatrist, therefore, must focus his attention on the patient's evaluation of the specific act, and not on his general ethical philosophy.

*Intent, Premeditation and Wilfulness.*—Usually a crime requires an overt act plus a specific intent, generally an intent to do harm.[32] Some crimes also require an element of premeditation. This offers another possibility for psychiatric analysis, since the physician may find that the defendant knew that the act was wrong, but that by reason of drunkenness, psychosis or deficiency he was unable to form the specific intent. For example, the law presumes that murder is in the second degree and imposes on the state the burden of proving the wilfulness and premeditation necessary to establish a murder in the first degree. The mental state of the defendant might be such that the prosecution could not prove the premeditation or deliberateness essential to the more serious finding; yet the defense would have to concede that the accused knew the nature, quality and wrongfulness of the homicide he had perpetrated. The net result is a sort of partial responsibility. Indeed, if, as lawyers are prone to do, the word "responsibility" is equated with "sanity," the effect is to establish "partial insanity," an admitted psychiatric absurdity, yet one which is logical enough for forensic purposes. In this connection, Glueck [33] stated: "The courts of at least two states have had sufficient vision to cope with the problem of the semi-irresponsible." The states referred to are Utah and New Jersey. In Utah, a court [34] has said: "A person's mental condition may not be such as to make him irresponsible, yet it may be such as to relieve him from the supreme penalty." The New Jersey decision [35] reads: "If the defendant was so feebleminded as to be incapable of forming the specific intent to kill with its wilful and deliberate character, then his offense would be murder in the second degree."

The physician should understand this concept clearly, since a man's life may depend on it. The psychiatrist is not finished when he decides simply that the accused is or is not psychotic. He must be prepared to say whether the defendant's mental state was such that he was capable of having the degree of intent, wilfulness, deliberateness or premeditation which the law might require for the offense. This is

32. *London* v. *Officer,* 242 Pac. 989.

33. Glueck, S.: Mental Disorder and the Criminal Law, Boston, Little, Brown & Company, 1925, p. 203.

34. *State* v. *Anselmo,* 148 Pac. 1071.

35. *State* v. *Schilling,* 112 Atl. 400.

particularly true of crimes committed by psychopathic persons during rages or by drunkards during periods of acute intoxication.

*Drunkenness.*—As a general rule, a state of alcoholic intoxication is no defense to a crime committed while in that condition, except when a man was made drunk against his will and by force. Such cases must be rare. A nice question is presented in a case of chronic alcoholic psychosis. In such a case the prosecution would admit that the crime was a result of an insane delusion, but would argue that since the insanity was the result of voluntary indulgence in alcohol the defendant was fully responsible. Most courts would probably exculpate the defendant, however, on the theory that his insanity was not a natural result (but a rather unusual result) of his drinking and that in any event he did not wilfully become insane. The psychiatrist's job would be simply to tell the jury that the accused had a psychosis, that it was due to alcohol and that it was a form of insanity.

Drunkenness sometimes robs a crime of one of its elements and thus reduces it to a lesser offense. If a drunkard assaults another man and inflicts grievous bodily harm, he may be indicted for assault with intent to kill or assault with intent to commit mayhem. The psychiatrist might be able to point out that the defendant was so befuddled by liquor that he could not form a clear intent to do anything. If the jury accepts that, the effect would be to reduce the crime to simple assault. Particularly difficult questions are raised by a crime committed during a period of pathologic intoxication. The patient has complete amnesia for his behavior and will never remember having done what he is charged with. But within the framework of his pathologic intoxication he will have acted sanely. He will show premeditation, planning and an effort to escape—all of which suggests that he has full responsibility even though his amnesia is genuine. In delirium tremens, on the other hand, the patient is clearly in no condition to plan, deliberate, weigh consequences, evaluate the wrongfulness of the act or understand its quality.

Some offenses are actually aggravated if the defendant is drunk (reckless driving, for instance), while others may be mitigated provided the psychiatrist satisfies first himself (and later the jury) that the intoxication stripped away one of the mental components of the act (such as intent or premeditation). Courts are not prepared, however, to accept the thesis that acute alcoholism is a disease. To the judges and the jurymen, intoxication is a bad habit and alcoholism a defect of character. The "drunk" usually knows he is doing something wrong. At the moment, however, he does not care.

*Irresistible Impulse.*—In most states, irresistible impulse is not a defense. A psychasthenic patient with pyromania is guilty of arson in spite of what the psychiatrist says. So is a man who "sees red" and flies into a rage during which he commits an assault.

Irresistible impulse, however, is accepted as a defense in about a dozen states. In Delaware,[36] the Supreme Court has spoken of "deprivation of will power to choose whether to do the act or refrain from doing it," and the Supreme Court of Alabama [37] has indicated that a defendant might be irresponsible if "by reason of mental disease he had so far lost the power to choose between right and wrong and avoid doing the act that his free agency was destroyed." Both these decisions, in effect if not in intent, provide a legal basis for pleading irresistible impulse. So does the military law, since the relevant phrase [38] in the "Manual for Courts-Martial" reads, "A person is not mentally responsible unless he was so free from mental derangement as to be able concerning the particular acts charged both to distinguish right from wrong and to adhere to the right." The last phrase, "to adhere to the right," certainly spells out a defense of irresistible impulse. The psychiatrist could testify with clear conscience, if his patient had genuine kleptomania, that the defendant knew that it was not right to steal but that because of his mental derangement (psychasthenia) he could not "adhere to the right—" or, in the language of the Alabama courts,[35] that "his free agency was destroyed," or, using the Delaware court's criterion,[36] that he was deprived of "will power to choose whether to do the act or refrain from doing it."

Professor Glueck [39] has examined decisions of appellate courts in every state in the union and cites cases showing that irresistible impulse has been accepted as a defense (at least once in each state) in Arkansas,[40] Colorado,[41] Connecticut,[42] Indiana,[43] Kentucky,[44] Michigan,[45] Ohio,[46] Pennsylvania [47] and Virginia,[48] in addition to Delaware [36] and Alabama,[37] as already indicated. This does not mean, of course, that the doctrine is firmly established in those states, but is simply a statement that in the cases cited the courts felt that the defendant should not be held accountable. Most of the states (certainly at least thirty of them) will not accept the defense of "irresistible impulse" and show little disposi-

36. *State* v. *Jack*, 58 Atl. 833.
37. *Parsons* v. *State*, 2 So. 854.
38. A Manual for Courts-Martial, reprint corrected to include the 1928 circulars. Document 14a, United States War Department, Office of the Judge Advocate General. Washington, D. C., 1928, p. 63, para. 78a, subpara. 6.
39. Glueck,[33] pp. 267-273.
40. *Williams* v. *State*, 50 Ark. 511 (1888).
41. *Ryan* v. *People*, 60 Colo. 425 (1915).
42. *State* v. *Saxon*, 87 Conn. 5 (1913).
43. *Morgan* v. *State*, 130 N. E. 528 (1921).
44. *Hall* v. *Commonwealth*, 155 Ky. 541 (1913).
45. *People* v. *Quimby*, 134 Mich. 625 (1903).
46. *Blackburn* v. *State*, 23 Ohio 146 (1872).
47. *Commonwealth* v. *Demarzo*, 223 Pa. 573 (1909).
48. *Thurman* v. *Commonwealth*, 107 Va. 912 (1908).

tion to change their doctrine, no matter how persuasive the psychiatric testimony in individual cases.

*Mental Deficiency.*—The McNaghten formula applies to mental deficiency, just as to the psychoses. Indeed, lawyers still confuse the two conditions. As recently as 1921, one high court said that "deficiency of intellect is a species of insanity." [35] The chief difficulty in applying the formula to mentally defective persons comes when the adult has a mental age in the 6 to 12 year bracket. Here, many psychiatrists reason that an adult with a mental age of, say, 10 years, ought to be judged with the same liberalness as that which would be applied to a 10 year old child. When the accused is a child, the prosecution always has to prove mental capacity to commit the crime. By contrast, if the accused is an adult, the capacity is assumed, and the defense has to prove lack of capacity if this is pleaded. Glueck [49] contends that "if it were shown that an adult is below the mental age of fourteen, it would seem more in consonance with the law to place the burden of proving mental capacity on the prosecution." The courts have not accepted Professor Glueck's recommendation. Thus, in 1 case it was held [35] that "when a man reaches maturity, the presumption is that he possesses capacity [to commit a crime] and it is for him to overcome the presumption." This is the general rule in all states.

Psychiatrists often introduce the defendant's mental age into the record. Judges and juries seem strangely unimpressed with the statement that the adult defendant really has the mind of a child. "Criminal responsibility does not depend on mental age," said a Massachusetts court [50] bluntly. Another Eastern court [51] expressed irritation with the whole thesis by saying that "the mental age theory is utterly misleading to a layman and practicaly useless in the administration of justice." An Arkansas judge [52] had this to say, "When an adult has the intelligence of a child of 7, that fact alone cannot be made the test as to whether he is or is not capable of committing a crime."

Intelligence tests, however, are useful in two particulars: (1) in helping to determine whether the defendant had sufficient wit to form the required degree of intent or deliberation (see paragraph on "intent") and (2) in assisting the judge in making disposition of a convicted defendant.

*Amnesia and Double Personality.*—A few years ago a common defense was, "Everything suddenly went black." More recently this has been replaced by the defense of simple amnesia. "I don't remember doing any of those things. If they say I did them, I guess I did; but

49. Glueck,[33] p. 196.
50. *Commonwealth* v. *Stewart,* 151 N. W. 74.
51. *State* v. *Ehlers,* 119 Atl. 15.
52. *Chriswell* v. *State,* 283 S. W. 981.

I have no recollection of it." Then, when it is demonstrated beyond doubt that the accused did do "those things," the explanation is that he had a double personality, on the Jekyll-Hyde pattern.

With reference to its legal status, the situation of the double personality seems to be this: Conceding that the patient had two personalities—one, the "main" personality, was good; the other, the "secondary" personality, was evil—the offense is now judged within the framework of the secondary personality, and the responsibility is then assigned to the main personality. Two illustrations may be cited.

A man habitually exhibited aggressive or suicidal reactions after he had only "a little" to drink. He invariably had a total (and genuine) amnesia for his actions during this state of pathologic intoxication. In one of these periods he attempted to rape a child. When she screamed, the parents came running out of the house, and the assailant jumped a fence and fled. He displayed great ingenuity in escaping capture, gained his own home and went to bed. He was awakened by police officers, seemed genuinely puzzled by the charge and insisted that he had been home all the time. In spite of positive identification, he would not allow his attorney to introduce any defense of intoxication. He kept repeating in a hurt and bewildered tone that he had not been near the scene of the crime. In this case, the question of responsibility may be considered within the framework of the intoxication, using the McNaghten formula. Since the patient tried to flee and carefully eluded capture, it is apparent that during that period he was conscious that he had done something wrong. The secondary personality thus appears to be criminally responsible. Projecting this responsibility on the main personality, one is forced to conclude that that, too, was responsible.

During the middle of the night, a soldier in a barracks dormitory got up and noisily proceeded to the clothes rack. He walked so carelessly that he awakened several other soldiers in the room. He inserted his hand into the pocket of another man's trousers. The other man got up, restrained him and reported the incident. The accused seemed confused and could say only that he was asleep all the time. His record of past conduct was perfect except for a verified medical history of somnambulism. Apparently, the somnambulistic personality had no consciousness of doing anything wrong, since he made no effort to walk silently. His behavior was designed, in effect, to facilitate capture, not to elude it. This secondary personality had no knowledge of doing anything wrong; and this lack of knowledge was associated with a nervous disorder, i. e., somnambulism. Under the McNaghten formula, the secondary personality was irresponsible. Therefore, under the rule enunciated, the main personality (i. e., the soldier himself) was not responsible.

Faced with a plea of amnesia or double personality, what does the psychiatrist do? He remembers that there are six possible explanations and explores all six of them. Except for some rare or freakish factor, the six explanations for this defense are (1) hysteria, (2) psychosis, (3) alcoholism, (4) head injury, (5) epileptic fugue or (6) the possibility that the defendant is telling a lie, made up out of whole cloth. Hysteria, if present, should be reflected in the total life pattern of the patient. It is also essential to decide whether the sequence of events was (*a*) first a hysterical fugue, then an offense committed while in

this trance, or (*b*) first the offense, then a hysterical wiping out of any memory of it. In situation *b* the patient would certainly be responsible. In situation *a* the responsibility would have to be adjudged by the McNaghten formula within the framework of the fugue. Amnesia associated with alcoholism takes one of two forms: ordinary drunkenness or pathologic intoxication. Drunkenness is evaluated as suggested in the paragraph on that subject. Pathologic intoxication is appraised in terms of the McNaghten formula, as applied to the secondary personality. Head injury is a possibility that must not be forgotten. Crimes are often associated with violence to the perpetrator as well as to the victim. An electroencephalogram may be helpful in diagnosis. With respect to behavior during a concussion-induced fugue, the description of the patient's activities should indicate whether he knew what he was doing or whether he really acted automatically; whether he acted as though he felt he were doing something wrong or whether he acted reflexly. Epileptic fugues should be diagnosable by the characteristic electroencephalographic tracing, as well as by an authenticated history of previous fugue states, trances or grand mal attacks.

In all cases in which amnesia or double personality is alleged, the psychiatrist will want to order an electroencephalographic examination and will search the past history for evidence of epileptic attacks, head injury, alcoholic habits and hysterical behavior.

*Presumption of Continuity of a Psychosis.*—Once it has been established that a person has a psychosis, it is presumed to continue. Discharge from a psychiatric hospital does not automatically adjudicate a return to sanity; it may mean only that the patient no longer needs custodial care or that he cannot profit from further treatment. The ex-inmate of a psychiatric institution is in good position to plead irresponsibility. Such a plea can be refuted, of course; but the point is that the prosecution has to carry that burden of proof. The state can show the hospital discharge, the limited nature of the psychosis and the fact that the patient had lately been conducting himself in a normal manner, all of which might well add up to proof of recovered sanity. Thus, in one case [53] the defendant committed a murder two years after discharge from a state hospital. The trial judge ruled (erroneously) that the defendant had to prove a psychosis if that was his plea. Apparently, he could not prove it, and he was convicted. The sentence was reversed by the upper court on the theory that his psychosis, established by his original commitment, must be presumed to continue, and that, if recovery is alleged, it is up to the prosecution to prove the recovery, not up to the defense to prove the psychosis.

53. *Davidson* v. *State*, 4 S. W. (2d) 74, Texas.

While a previous psychosis is presumed to continue, present insanity will not be historically projected backward. If a defendant acquires a psychosis in jail awating trial, this fact has no value in proving that he was psychotic at the time of the crime. As an Alabama court [54] put it, "The Supreme Court knows of no rule making the establishment of insanity a presumption that the party was insane prior thereto."

When no psychosis is established by past history, the presumption is that the accused is sane, no matter how fantastic the crime. If the defendant alleges irresponsibility, he must prove it. In some states he must prove it "beyond a reasonable doubt"; in others, "by a preponderance of the evidence," and in still others, simply "to the satisfaction of the jury." The degree of proof has some bearing on psychiatric testimony. For instance, one good psychiatrist says the accused is psychotic and another says that he is sane. The jury might happen to feel that the former practitioner was more of an expert or had had better chances to know the defendant than the latter. If "to the satisfaction of the jury" or "by a preponderance of the evidence" is the measure, the defendant would be found not guilty. But if irresponsibility had to be proved "beyond a reasonable doubt" then the proof here would fall short, since if the two experts are so evenly balanced the proof is scarcely "beyond a reasonable doubt."

*The Psychiatrist's Role.*—Sent to examine a defendant, the psychiatrist should be certain that he is not unfairly trapping the patient into making damaging confidential admissions to a physician which would later be used against him. Legally, the psychiatrist is on solid ground, since submitting to a mental examination is not considered compulsory self incrimination. No person can be compelled to give testimony against himself. If the result of the mental examination is unfavorable to the defense, it might be argued that the psychiatric interview was, in effect, an underhand way of getting the patient to incriminate himself. There is no legal substance to this thesis, but physicians should err on the side of ethical scrupulousness and be sure that there can be no moral basis for it, either. On the legal side, the following two dictums are illuminating:

> Testimony showed that the doctor informed the accused that he had been requested by the county attorney to make the examination. He told the accused that he did not have to answer any questions. The accused submitted to the mental tests without objection. Such testimony is admissible when accused submits to an examination if there were no threats, duress, objection or deceptions. Such an examination is not subject to the objection that the defendant was compelled to give testimony against himself.[55]

54. *Birchfield* v. *State*, 115 So. 297.
55. *Wehenkel* v. *State*, 218 N. W. 137 (Neb.).

An examination of the defendant to ascertain mental condition is not a deprivation of constitutional rights; nor does such an examination require him to give evidence against himself.[56]

While the psychiatrist is thus legally safe in conducting an examination without consent of (or knowledge of) the defense attorney, it is good form for him to apprise the patient that he is taking notes, that he is going to submit a report to the court (or prosecutor, or probation officer) and that the patient does not have to tell him anything. My own practice is to say: "I'm a doctor, trying to understand how you happened to get into this trouble. You're under no obligation to answer my questions or tell me anything, but I do hope that whatever you do tell me will be the truth. If you'd rather not answer a question, just say so; and it will be perfectly all right. I'm making notes, and afterwards I'll send in a report." It might be expected that such a warning would dry up the patient's flow of words, but it usually has no such effect. At least, after the defendant "warms up" to the subject, he generally talks freely enough.

It is essential that the psychiatrist take elaborate notes and that large fragments of the patient's remarks be written down verbatim—not that such comments have to be included in the report, but, rather, that the examiner may be in a position to support his conclusions by first hand evidence. In Britain [57] it is considered "not cricket" for the physician to ask the accused whether he has committeed the crime, but American psychiatrists often find it impossible to conduct a thorough mental examination without somehow touching on that point. The general technic of the examination differs little from the routine of a psychiatric examination made elsewhere except that the physician must keep the four elements of the McNaghten formula clearly in mind. Did the defendant know the nature of the act? The quality of the act? The wrongfulness of the act? And, if so, was this because of a mental disorder?

It is unwise to conduct this kind of mental examination without a preliminary briefing. The psychiatrist should get the facts from the investigating officer and should ask permission to read the statements of the witnesses and of the accused. For instance, a crime may have been committed in a rage. The witnesses' stories of how the patient

56. *Blocker* v. *State*, 110 So. 547 (Fla.).

57. For instance, on page 233, Mercier [28] warns "the examiner against questioning a prisoner as to whether or not he had committed the offense or as to the circumstances of the crime. I have heard caustic comments from the bench on this practice." Apparently, the British psychiatrist is not expected even to review the surrounding circumstances, let alone the question of guilt. I cannot see how the physician can evaluate the patient's understanding of the particular offense if the interview is subject to this kind of limitation.

looked, what he said, how he acted, are helpful to the examiner in determining whether this mental state was an epileptic furor, a drunken rage, or a momentary lapse of inhibition in an otherwise normal man.

The psychiatrist's next duty is to make up his own mind. Courts are impatient with an examiner whose conclusion is that "in a way he was responsible; yet in another sense he wasn't." True, in most cases, it is not simply a matter of black or white. Still, the courts want help, and the psychiatrist is presumably more of an expert on mental states than any one else. A cleancut, crisp, concrete opinion is expected. As guideposts to reaching a definite decision, the psychiatrist can remember the doctrines with reference to irresistible impulse, alcoholism, mental age, amnesia and the basic McNaghten formula, elaborated earlier in this paper. These provide both a framework around which the examination may be constructed and a battery of criteria by which responsibility may be evaluated.

Actual decision as to the defendant's responsibilty is not made by the psychiatrist. It is the function of the jury, or in special cases the prerogative of the judge of the trial or the appellate court. The practitioner does not testify that the defendant was responsible or irresponsible. That would be a usurpation of the jury's function. He testifies as to the clinical diagnosis and as to his opinion of the patient's intent and deliberateness and of his realization of the nature, quality and wrongfulness of his acts. There the psychiatrist's responsibility ends.

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# Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

## Anatomy and Embryology

THE ROLE OF THE SENSORY FIBERS IN THE DIFFERENTIATION OF THE SPINAL CORD IN SHEEP. DONALD H. BARRON, J. Exper. Zool. **100**:431 (Dec.) 1945.

Barron amputated the distal segments of the forelimb in 4 sheep fetuses in utero after their fifty-fifth day of development. It has been shown that at this stage of development mitosis in the anterior cervical and brachial segments of the spinal cord and the associated ganglia has ceased.

Except for the structures associated with remnants of the amputated limbs, the 4 fetuses, recovered near term by cesarian section, appeared to have developed normally.

The individual spinal ganglia of the affected brachial region were reduced in volume and number of neurons in proportion to the extent to which their distal processes normally supplied the limb. The number of fibers extending into the gray matter by the individual dorsal roots of the affected side were in proportion to the number of neurons in the ganglion of origin. When the reduction in volume of gray matter and number of neurons was most pronounced, there was a hypoplasia of neurons of 34 per cent in the substantia gelatinosa and of 13 per cent in the intermediate gray matter of the seventh cervical segment. This hypoplasia on the affected side appears to be the result of the failure of the normal complement of fibers to enter these regions.

The hypothesis that the growing processes of neuroblasts induce indifferent cells of the nervous system to differentiate is supported, although not proved, by these observations.

REID, Boston.

THE DEVELOPMENT OF LATE GASTRULA EXPLANTS OF RANA PIPIENS IN SALT SOLUTION. HARRY S. EMERSON, J. Exper. Zool. **100**:497 (Dec.) 1945.

Explants of eight areas from the oval blastopore stage were transplanted into full strength bicarbonate-free Holtfreter salt solution (*Arch. f. Entwicklungsmechn. d. Organ.* **138**:657, 1938). In contrast to other areas, explants of areas 1 and 2 often disintegrated, but all those which overcame the immediate toxic effect lived as long as the initial yolk content of the cells persisted.

The results show that area 1 is presumptive chordamesoderm, areas 2 and 3 are presumptive neural plate, areas 4 through 7 are epidermis and area 8 is presumptive lateral plate mesoderm.

Unlike the behavior of gastrula transplants in regenerating tissue, horny jaws, nasal sacs and lenses come only from explants from the presumptive locations of these organs.

Brain and eyes develop about as well from isolates of ectoderm with as from those without the substratum, while horny jaws and nasal sacs require ectoderm plus substratum. Apparently, horny jaws as well as nasal sacs and neural plate are induced by some part of the archenteron roof. These three organs appear to be induced by different parts of the chordamesoderm acting on the ectoderm at different times during gastrulation.

REID, Boston.

THE DEVELOPMENTAL RELATIONSHIP BETWEEN THE NERVOUS AND EPITHELIAL COMPONENTS OF THE HYPOPHYSIS. ERLING S. HEGRE, J. Exper. Zool. **101**:65 (Feb.) 1946.

Hegre made observations on the growth and development of the epithelial rudiment of the hypophysis when grafted by itself or together with tissue from the

diencephalic floor. The tissue was transplanted from Amblystoma maculatum embryos (Harrison's stage 33) into the sinus venosus of hosts of the same species (stage 36).

There were three groups of host animals: (1) hosts grafted with the whole pituitary gland; (2) hosts grafted with the epithelial rudiment with a few cells of the diencephalon attached, and (3) hosts grafted with the epithelial rudiment only.

Exposure of the host animals to a light background was used as a test for the presence of a pars intermedia in the developing grafts.

A pars intermedia developed in every case in which the whole pituitary gland was implanted. The sectioned grafts showed typical pituitary tissue, together with recognizable nerve tissue. A pars intermedia developed in 55 per cent of the implants of group 2, and nerve tissue was always found in direct association with this portion of the graft. Pituitary tissue was recovered from 96 per cent of grafts of group 2.

No pars intermedia developed when only the epithelial rudiment was transplanted, although pituitary tissue was recovered from 89 per cent of the animals sectioned.

The author concludes that the presence of nerve tissue (diencephalic floor) is necessary for the formation of a pars intermedia from the epithelial hypophysis in Amblystoma maculatum.

REID, Boston.

## Physiology and Biochemistry

DEGREE AND NATURE OF REGENERATION OF SPLANCHNIC INNERVATION TO THE ADRENAL GLAND TWO YEARS FOLLOWING COMPLETE BILATERAL SYMPATHECTOMY IN DOGS. J. W. PAPEZ, A. V. JENSEN and H. H. DUKES, J. Neurophysiol. **8**:1 (Jan.) 1945.

Papez, Jensen and Dukes removed the thoracolumbar portion of the sympathetic chain with the rami communicantes and the splanchnic nerve bilaterally in 2 dogs. No alterations of behavior occurred after the operation. Autopsy two years later showed pseudoregeneration of the sympathetic trunk on one side in 1 animal and bilaterally in the other. These false nerve trunks consisted chiefly of perineurial strands, with only occasional regenerated nerve fibers. The nerve fibers not only were few but were also distorted, and not more than 5 per cent pursued a course to the medulla of the adrenal gland. The authors observed postganglionic vasomotor nerves of moderate amount coming from the celiac and periadrenal ganglia, and these fibers were not affected by section. In a third dog, killed fourteen days after sympathectomy, denervation occurred on one side, and on the other the fibers were in stages of fragmentation and degeneration.

Papez, Jensen and Dukes conclude that regeneration of splanchnic and sympathetic nerves after total sympathectomy is small and that the pseudoregenerated trunks contain few functioning fibers.

FORSTER, Philadelphia.

RESTORATION OF VISION AFTER CROSSINGS OF OPTIC NERVES AND AFTER CONTRALATERAL TRANSPLANTATION OF THE EYE. R. W. SPERRY, J. Neurophysiol. **8**:15 (Jan.) 1945.

Sperry studied the effects of crossing of the optic nerves in 21 anurans and of contralateral transplantation of the eye in 2 anurans and 12 urodeles. In all experiments intrinsically well organized visuomotor coordinations were recovered. The recovered visuomotor responses, however, were maladapted and were schematically correlated with the anatomic rearrangements. The intrinsic organization, therefore, did not arise from learning but was a product of the process of regeneration. The reestablishment of functional relation between retina and visual centers, therefore, is predetermined by growth-regulating factors and not by functional adaptation.

FORSTER, Philadelphia.

OBSERVATIONS OF GAS BUBBLES IN PIAL VESSELS OF CATS FOLLOWING RAPID DECOMPRESSION FROM HIGH PRESSURE ATMOSPHERES. CARRELL E. WAGNER, J. Neurophysiol. **8**:29 (Jan.) 1945.

Wagner observed through a Forbes window the pial vessels of cats subjected to compression for one hour, followed by rapid decompression. Gas bubbles appeared in pial arteries before they did in pial veins; the distribution of gas bubbles in other vessels was independent of their appearance in pial vessels. The author concludes that gas bubbles are borne to their site of lodgment as air emboli. The appearance of gas bubbles in the veins is secondary to the decrease in the blood flow through the region.

FORSTER, Philadelphia.

PROPAGATION OF SPREADING CORTICAL DEPRESSION. A. P. LEAO and R. S. MORRISON, J. Neurophysiol. **8**:33 (Jan.) 1945.

Leao and Morrison studied the depression of the cerebral cortex of rabbits induced by electrical stimulation. Spreading depression could be elicited from all areas studied except area Rsg of Rose. When depression was induced in one hemisphere spread to the opposite hemisphere was found to depend on the integrity of the corpus callosum. Spread of the depression was independent of subcortical connections and of the three lower laminas of the cortex. Acute anemia of the cortex lasting up to one minute did not interfere with the spread of depression. Depressions were elicited by short weak tetanic stimulation of the surface of the cortex and stimulation with direct current. An isotonic solution of calcium or potassium was most effective in eliciting the depression. Physostigmine, acetylcholine, strychnine, cocaine, histamine and epinephrine were ineffective. Applications of cocaine to the cortex succeeded in restricting the spread of depression and vasodilatation. Leao and Morrison do not believe that the phenomenon depends on a neuron to neuron transmission of inhibitory impulses.

FORSTER, Philadelphia.

INFLUENCE OF DEPLETION OF DIFFUSIBLE ELECTROLYTES UPON ELECTRICAL ACTIVITY OF THE BRAIN. ERNST P. PICK and MICHAEL M. MILLER, J. Neurophysiol. **8**:47 (Jan.) 1945.

By immersing frogs in distilled water, Pick and Miller altered the selective permeability of the frog's skin. In this manner they depleted the diffusible electrolytes. Study of the electrical activity of the cortex of the frogs revealed that cortical activity was decreased and that either slow waves appeared or both the frequency and the amplitude of the waves were decreased. The degree of reduction varied with different cortical regions. Unless the electrical activity has been too seriously impaired by loss of electrolytes, the restoration of chlorides results in return of normal electrical activity of the cortex. The depression of electrical activity is considered to be due to the inhibitory effects on oxidative processes of the brain during deprivation of chloride and glucose. Pick and Miller suggest that the successful treatment of shock with saline solution may be due in part to the central action of the saline solution.

FORSTER, Philadelphia.

NERVE REGENERATION IN CATS ON VITAMIN $B_1$-DEFICIENT DIETS. CHARLES BERRY, CHARLES NEUMANN and JOSEPH C. HINSEY, J. Neurophysiol. **8**:315 (Sept.) 1945.

Berry, Neumann and Hinsey found that a raw carp diet produced in cats anorexia, ataxia, vestibular disturbances and convulsions. A better controlled tube-fed, thiamine-deficient diet permitted survival up to one hundred and sixteen days, with only mild ataxia and vestibular signs. Peripheral nerves excised immediately after death from thiamine deficiency were found to conduct impulses, as shown in recordings of action potentials. Microscopic examination of the nerves revealed no degeneration. The tibial, peroneal and saphenous nerves regenerated normally

in the presence of severe thiamine deficiency. Regeneration was determined by growth of the fibers longitudinally and in diameter and by recording the action potentials of the nerves.

FORSTER, Philadelphia.

CARBONIC ANHYDRASE IN THE NERVOUS SYSTEM. HORACE W. DAVENPORT, J. Neurophysiol. **9**:41 (Jan.) 1946.

Davenport found that thiophene-2-sulfonamide in concentrations of 27 mg. per hundred cubic centimeters or higher had no effect on the rate of conduction of impulses, voltage capacity curves and recovery of responsiveness in peripheral nerves of cats, the spinal reflexes of decerebrated cats or the electrical activity of the cerebral cortex of anesthetized rabbits. Since carbonic anhydrase of the central nervous system is inhibited more than 99.99 per cent by these concentrations of thiophene-2-sulfonamide, Davenport concludes that catalyzed hydration and dehydration of carbon dioxide are not essential reactions in the nervous system.

FORSTER, Philadelphia.

## Neuropathology

A NEW ABSORBABLE MATERIAL FOR USE IN NEUROLOGICAL AND GENERAL SURGERY. E. B. SPITZ, M. ZIFF, C. BRENNER and C. DAVISON, Science **102**:621 (Dec. 14) 1945.

In preliminary experiments here reported, pledgets of "aralac" batting (a casein fiber from milk) (30 to 150 mg., total dry weight) were inserted into the subcutaneous tissue, the temporal muscle, the subdural and subarachnoid spaces and the substance of the brain itself in a series of 6 cats. Each operation was performed aseptically with the animal under sodium pentobarbital anesthesia, and the cats were allowed to survive for eleven, eleven, twelve, twenty-three, thirty-five and forty-six days, respectively. At the end of these periods the animals were anesthetized and the brains removed, along with other tissues that had been in contact with the batting. None of the animals showed any ill effects from the insertion of the casein fiber, and each was in good health at the termination of the experiment.

At autopsy, no casein fiber was visible grossly in any specimen. Microscopically small amounts of the fiber remained in the 3 animals that were examined at the end of eleven, eleven and twelve days, respectively. A few of the fibers were bordered with giant cells. The principal tissue reaction consisted of the presence of macrophages or, in a few instances, of polymorphonuclear leukocytes and lymphocytes, on the one hand, and proliferation of fibroblastic and endothelial cells, on the other. In the 3 cats which were allowed to survive the longest no trace of fibers remained. Giant cells were rarely seen in the sections from these animals. The tissue reaction consisted chiefly of proliferation of fibrous tissue and the presence of macrophages at or near the site of implantation. Two animals served as controls, and microscopic sections from these animals revealed that the tissue reaction was slightly greater on the side on which the casein fiber had been implanted. There was no evidence in any section that either the casein fiber itself or its breakdown products were particularly toxic to the cortical cells.

From these preliminary experiments, the authors conclude that casein fiber ("aralac") is rapidly absorbed after septic implantation in the brain, muscle or subcutaneous tissue of the cat. The absorption is accompanied with a mild cellular reaction, similar to that produced by implantation of fibrin foam or oxidized cotton.

GUTTMAN, Philadelphia.

NEUROPATHOLOGIC PROBLEMS AFTER PREFRONTAL LEUKOTOMY. A. MEYER and E. BECK, J. Ment. Sc. **91**:411 (Oct.) 1945.

The material reviewed by Meyer and Beck consists of 7 cases of prefrontal leukotomy and an eighth case in which frontal lobectomy had been carried out on

a mentally normal person with a basal meningioma. The last case has proved of great value as a test case for complete severance of fiber connections. In 2 additional cases only macroscopic investigation was performed. All the patients who recovered showed little or moderate deterioration. Clinical complications did not interfere with recovery, and the operation itself produced circumscribed lesions uncomplicated by severe hemorrhage and softening. Of the 3 patients of the unrecovered group, 1 (case 5) was severely deteriorated. The 2 others (cases 3 and 4) were little deteriorated and should have had a fair chance of recovery. However, in case 3 uremia, nicotinic acid deficiency and a senile condition of the brain must be considered as adverse factors. In case 4 the cut was placed too posteriorly, involving the agranular frontal cortex and the caudate nucleus. In addition, extensive hemorrhage had destroyed a considerable part of the white matter of the prefrontal lobe. In case 5 the cut was placed in too anterior a plane. As regards the recovered group, it is surprising how little damage was sufficient to produce a remarkable clinical improvement. The section in these cases was incomplete with regard to severance of the thalamofrontal fibers. From these results it follows that in suitable cases (in which deterioration has not occurred) a good recovery may follow surprisingly incomplete and circumscribed cuts in the white matter of the prefrontal lobe. Recovery may be prevented by an excessive lesion, either by cutting in too posterior a plane or by causing extensive hemorrhage. The avoidance of hemorrhage seems to be a major necessity. There is some evidence that the projection of thalamic fibers to the orbital region is of considerable strength and that the cortex in front of the surgical lesion is not wholly unaffected. The centrum medianum (of the medial nucleus of the thalamus) displays in the human brain a considerable variation of cell density. Its significance requires further investigation. The only conclusion that can be drawn at present is that under both normal and psychotic conditions the centrum medianum is liable to considerable variations in cell density and spacing of cells, which apparently far exceed the variability in animal brains. This variability is of remarkable interest if one regards the nucleus as an association center within the thalamus, which itself appears to be one of the cornerstones of what one might call the anatomic substrate of personality organization. It is the more remarkable, as previous work on individual architectural differences within the cerebral cortex has demonstrated the relative constancy of the cortical pattern.

J. A. M. A.

## Psychiatry and Psychopathology

HEREDITY IN THE FUNCTIONAL PSYCHOSES. RALPH ROSENBERG, Am. J. Psychiat. **101**:157 (Sept.) 1944.

Rosanoff studied the role of heredity as an etiologic factor in schizophrenia by its incidence in twins. Of 41 pairs of identical twins, schizophrenia appeared in both members in 68 per cent, and in 32 per cent of the pairs only 1 twin had the disease, indicating the absence of a hereditary basis. In another group (same sex; twins not identical), schizophrenia was present in both twins in only 20 per cent. In the third group (opposite sex; twins not identical), schizophrenia occurred in both twins in only 10 per cent.

Since hereditary relations are the same in the three groups, the difference between twins of the same sex, twins of the opposite sex, and ordinary siblings is a definite measure of environmental differences. Environment, and not heredity, is the vital factor in the development of functional psychoses, as shown by Rosenberg.

BORKOWSKI, Philadelphia.

AFFECTS, PERSONAL AND SOCIAL. GREGORY ZILBOORG, Psychoanalyt. Quart. **14**:28, 1945.

There have been, and continue to be, disagreements between the sociologist, the culturalist and the psychoanalyst as to the basis of social psychology. The sociologist and the culturalist reject psychoanalysis as inadequate because it seems to

lack a comprehensive social theory. The psychoanalyst, transposing the general theory of the neuroses onto the field of sociology—where it really is not applicable—decries the sociologist and the culturalist.

Zilboorg states that affects are the best indicators of the forces which are involved in a given aspect of behavior, and he proceeds to contrast the individual affects in the neuroses, in mobs and in social feelings. There are certain outstanding characteristics of social feelings. Man considers his opinions on public issues his own, and in this he differs from a member of a mob. Social affect and behavior are not impulsive, but are stable, steady and marked by an enduring sense of loyalty, particularly toward a specific group within the social whole. This loyalty is combative toward other specific groups. The man who states his social feelings tends to idealize them and to formulate an allegedly rational and ethical ideology. The social group is based not on a solidarity born of love but on a cohesion based on hate of an "external" enemy, supposedly always present and ready to strike.

The determinants for these affects are not purely libidinal in origin. They are cultural economic determinants which utilize the libidinal equipment for the special purpose of a given group. Although the social affects are marked by hate, they also contain love, which in the social affects appears almost impersonal—it is directed toward a remote, invisible goal, i. e., toward a fantasy. Basically, social affects are based on the gratification of pregenital libidinal drives, which all of us are personally attempting to thwart but which can be gratified in the permissive setting afforded by our culture.

PEARSON, Philadelphia.

REPATRIATION: A PSYCHIATRIC STUDY OF 100 NAVAL EX-PRISONERS OF WAR. W. P. MALLINSON and W. WARREN, Brit. M. J. **2**:798 (Dec. 8) 1945.

Mallinson and Warren made a psychiatric study of 100 naval ex-prisoners of war in an effort to obtain a clearer picture of the process of resettlement and of the normal reactions following repatriation. Of this number, 71 were apparently making a satisfactory readjustment and 29 exhibited neuropsychiatric symptoms. In the group of 29 patients with abnormal reactions, 25 showed only minor neurotic reactions, and 4 had disorders of sufficient severity to necessitate hospitalization or invalidation. The patients in the abnormal group complained of the usual symptoms of the minor affective disorders, frequently associated with psychosomatic complaints and with restlessness and irritability, and responded to the usual methods of treatment. The most important factor precipitating neurotic reactions was constitutional predisposition; psychogenic factors operating before, during and after captivity also played a part.

ECHOLS, New Orleans.

## Diseases of the Brain

NERVOUS SYSTEM DYSFUNCTION IN ADAPTATION TO HIGH ALTITUDE AND AS POSTFLIGHT REACTIONS. GORDON A. BROWN, CHARLES A. CRONICK, HURLEY T. MOTLEY, ELMER J. KOCOUR and WALTER O. KLINGMAN, War Med. **7**:157 (March) 1945.

This paper is a description of some of the dysfunctions of the nervous system observed in flying personnel of the Army Air Force during and after oxygen-controlled high altitude conditions.

In general, the clinical neurologic reactions fall into the following categories: (1) disturbances of equilibrium and coordination; (2) disturbances of function of large sensory or motor tracts; (3) disturbances of consciousness and cortical function in the nature of amnesia, confusion and disorientation; (4) disturbances suggestive of meningeal irritative phenomena, increased intracranial pressure and migraine-like attacks; (5) disturbances of subcortical mechanisms associated with phenomena of dyskinesia, hyperkinesia and aphasia, and (6) scattered disturbances of the nervous system of minor character.

The authors have classified the disturbances into these categories to emphasize that there seems no one part of the nervous system more resistant or vulnerable than another.

The dysfunction of the nervous system occurs not only during the period of great change in atmospheric pressure but after exposure to conditions of high altitude and return to the ground level, despite a constant oxygen supply. The symptoms are not immediately or shortly relieved by continued administration of oxygen.

Recovery in all cases was prompt and without detectable residual symptoms.

PEARSON, Philadelphia.

CRANIAL DEFECTS FOLLOWING CLOSED HEAD INJURY. T. ALAJOUANINE and R. THUREL, Rev. neurol. **77**:71 (March-April) 1945.

Alajouanine and Thurel report 4 of their own cases and cite 2 from the literature in which trauma to the head at birth or very early in life resulted in subsequent development of extensive defects in the cranium at the site of injury. As the skull grows and the brain expands, the site of injury to the bone becomes more extensive. The authors believe the cerebral lesions observed in many of these cases, resulting in hemiplegia and convulsions, are due to hernia of the brain rather than to the defect itself. In case 3 the hemiparesis came on eleven months after the injury, a few months after the loss of cranial substance was observed.

N. SAVITSKY, New York.

TUBERCULOMA OF THE CEREBRUM: OPERATION AND SURVIVAL FOR THREE YEARS. T. ALAJOUANINE and R. THUREL, Rev. neurol. **77**:209 (July-Aug.) 1945.

Jacksonian attacks appeared in a girl aged 17 in July 1941. In December 1941 neurologic examination revealed a normal condition. Lumbar puncture yielded normal readings. A pneumoencephalogram showed that the ventricles did not fill; markings on the left side of the brain were somewhat less definite than those on the right. After the insufflation of air there was a remission from the jacksonian attacks of two months. In February 1942 the attacks reappeared, with paralysis of the right upper extremity, hyperreflexia of the right arm and loss of position sense in the last three fingers of the right hand. A second aerogram showed indubitable evidence of an expanding lesion. The patient did not have tuberculosis, but her father died of intestinal tuberculosis and her paternal grandmother of tuberculosis of the spine. A roentgenogram of the chest showed nothing abnormal, and the reaction to tuberculin was negative. Operation, performed on March 6, 1942, revealed a tumor at a depth of 1 cm. in the left frontoparietal cortex. The tumor did not look grossly like a tuberculoma. It was well demarcated from the surrounding tissue of the brain but was difficult to separate from it. During August 1943, pulmonary tuberculosis developed, and in May 1945 she died of tuberculous meningitis. A pneumoencephalogram just before death showed dilatation of the ventricles, especially on the left side, but no evidence of recurrence of the tumor. This case shows that a cerebral tuberculoma can be removed without dissemination of the tubercle bacilli.

N. SAVITSKY, New York.

DIAGNOSIS AND SURGICAL THERAPY OF CEREBRAL MANIFESTATIONS OF THROMBOANGIITIS OBLITERANS (WINIWARTER-BUERGER DISEASE). H. KRAYENBÜHL, Schweiz. med. Wchnschr. **75**:1025 (Nov. 24) 1945.

Krayenbühl reports 25 cases of cerebral manifestations of thromboangiitis obliterans (Winiwarter-Buerger disease). In 3 of these cases the cerebral manifestations accompanied a more or less generalized thromboangiitis obliterans, while in 22 the thromboangiitis was restricted to the cerebral blood vessels. Careful cardiovascular examination and cerebral arteriography offer considerable aid in the diagnosis of the rare cases of the first group. Diagnosis proved more difficult in the second group. The age at which the initial symptoms of the disease

occurred varied from 11 to 58 years. Signs of unilateral motor or sensory paralysis of variable degree and extent in all the patients were revealed on examination or by the history. Ocular symptoms were rare. Temporary spasms of the vessels of the retina were not observed. A drop in arterial blood pressure of the retina was demonstrated occasionally. There were no symptoms of increased intracranial pressure. In 12 cases the complete occlusion or a more or less high degree of constriction of the internal carotid artery was demonstrated at the point where the common carotid artery gives off the internal carotid artery; this, therefore, is to be considered as the area of predilection of the vascular process. In 4 cases the occlusion of the internal carotid artery occurred in the carotid "siphon"; this term is applied to the point where the internal carotid artery gives off the middle cerebral artery.

J. A. M. A.

IMPAIRMENT OF PUPILLARY REACTIONS DURING ACCOMMODATION SUPERIMPOSED ON ARGYLL ROBERTSON PUPILS ASSOCIATED WITH TABES DURING ACUTE ANEMIA. BENJAMIN SPOTA and DIEGO BRAGE, Prensa méd. argent. **32**:1598 (Aug.) 1945.

Spota and Brage report 2 cases of tabes dorsalis with Argyll Robertson pupils, in both of which there was secondary anemia from bleeding hemorrhoids and ulcer of the stomach. There was pronounced impairment of pupillary reactions in accommodation during the period of the anemia. Another case of tabes with Argyll Robertson pupils is recorded in which the pupillary reactions during accommodation became impaired after exposure to carbon monoxide.

These facts indicate that the centers for pupillary reactions during accommodation are diseased to some extent in all cases of tabes. The defect in accommodation becomes clinically manifest when anything interferes with the proper oxygenation of this center.

N. SAVITSKY, New York.

## Treatment, Neurosurgery

PENICILLIN IN THE TREATMENT OF SYPHILIS. EDITORIAL, Ann. Int. Med. **26**:148 (Jan.) 1947.

Like many another new form of therapy, the penicillin treatment of syphilis has progressed through the cycle of dubiety, enthusiasm and reaction. Mahoney's original report that penicillin is effectual against Treponema pallidum was received with some caution, but as laboratory and clinical confirmation of this observation rapidly became available reserve quickly gave way to enthusiasm. So promising did this new form of therapy appear that in September 1943, within three months of the first public announcement, a nation-wide cooperative study was organized under the auspices of the Committee on Medical Research. In less than a year penicillin was adopted for routine use in cases of early syphilis by the United States Army. Since then the limitations of this form of therapy gradually have become apparent.

The principal advantages of penicillin in the treatment of syphilis are its lack of toxicity and the fact that the therapeutic schedule need not be inordinately prolonged. Consequently, the full course of treatment is almost invariably completed. This is not the case with any form of arsenotherapy, with which toxic reactions increase in frequency the more the total duration of treatment is compressed and with which case holding becomes increasingly difficult as the period of therapy is prolonged.

To be feasible as an agent for treatment of ambulatory patients with syphilis in the clinic and in the physician's office, a modified preparation of penicillin with prolonged activity is desirable. Many attempts have been made to extend the duration of action of penicillin, either by delaying its absorption or by blocking its excretion by the renal tubules; but by far the most satisfactory modification presently available is the suspension of penicillin in peanut oil and beeswax.

This preparation of penicillin has been used in the treatment of syphilis. Preliminary reports suggest that the results may be sufficiently satisfactory to warrant more widespread application. Treatment schedules utilizing this preparation alone and in combination with oxophenarsine hydrochloride or a bismuth compound are being evaluated currently by the clinics cooperating in the nationwide study of syphilis. Already there is some indication that with administration of as much as 9,600,000 units of penicillin in oil and beeswax over a period of sixteen days there is a not inconsiderable number of treatment failures.

With any schedule of administration of penicillin the data to date indicate that there has been a high incidence of treatment failures. After the Army adopted the schedule of administration of 2,400,000 units in seven and one-half days relapse rates have been several times as high as the rates after any schedule of arsenobismuth therapy, prolonged or intensive, provided that the latter was fully completed.

There is here involved the possibility that penicillin may actually be more efficacious in treatment of early syphilis than appears from this comparison, and that many so-called relapses actually represent reinfection. Unfortunately, this point is incapable of determination on the basis of existing clinical and experimental data.

It is believed that this excessively high incidence of treatment failures with penicillin may be reduced in two ways. The total duration of treatment may be prolonged, in which case there arises the problem of case holding, so frequently encountered during metal chemotherapy. Perhaps a more promising approach is the addition to the penicillin treatment of a scheme of concurrently administered metal chemotherapy.

There is no unanimity of opinion as to the desirability of combining penicillin and oxophenarsine hydrochloride in the routine treatment of early syphilis. Some authors have expressed the belief that the results with penicillin alone, when administered in adequate amounts over a long enough period, are satisfactory in a sufficiently large proportion of patients to justify eliminating arsenicals from the original course of treatment, reserving their use for patients with relapse. Others believe that the additional therapeutic effectiveness provided by arsenic warrants the increased risk.

In the management of neurosyphilis, penicillin is proving of significant worth. On the abnormalities of the cerebrospinal fluid, especially on the pleocytosis and the elevated protein content, which have been considered an indication of the "activity" of the process in the central nervous system (Dattner-Thomas), penicillin exerts a profoundly favorable effect. This is true not only with asymptomatic neurosyphilis but also with the various clinical syndromes of syphilis of the central nervous system, although post-treatment "reactivation" has been noted somewhat more frequently among patients with symptomatic (usually parenchymatous) neurosyphilis than among those with asymptomatic involvement of the neuraxis.

In cases of asymptomatic neurosyphilis, in which the only evidence of involvement of the central nervous system is an abnormal spinal fluid, the result of therapy can be adjudged only by the response of the spinal fluid and the incidence of progression to clinical neurosyphilis. The abnormalities of the spinal fluid in early and in late asymptomatic neurosyphilis respond dramatically to penicillin. Improvement is manifest promptly in the cell count and the protein content, more gradually in the colloidal gold curve, and, last of all, the Wassermann reaction. Normality of the spinal fluid, once achieved, seems usually to be stable. The rapidity with which the spinal fluid becomes normal after penicillin therapy is dependent on the degree of the pretreatment abnormalities and the duration of the syphilitic infection. Abnormalities of lesser degree and those occurring within the first two years of the disease disappear rapidly; those which are more extensive and of longer duration improve slowly over a period of years.

The ultimate result in terms of clinical progression will not be known for many years. If, however, the favorable responses of the spinal fluid thus far noted are sustained, the incidence of clinical neurosyphilis developing in this group of patients should be low.

The clinical manifestations of neurosyphilis are protean: Some are due to active inflammation and others to degenerative processes; some are reversible, and others are the result of irreparable damage of neural tissues. In its effects on these clinical manifestations, which include such widely dissimilar symptom complexes as acute syphilitic meningitis, dementia paralytica, tabes dorsalis and Erb's spastic paraplegia, the presently available information suggests that penicillin is superior to metal chemotherapy but that it gives little promise of clinical results in treatment of parenchymatous neurosyphilis superior to those obtainable with fever therapy.

It should be pointed out, however, that such improvement as does follow penicillin therapy is attained at no risk to the patient, and is obtained in a shorter time and with less inconvenience to him than attends either fever therapy or protracted metal chemotherapy.

With acute syphilitic meningitis the results of therapy with penicillin used alone are excellent, but with parenchymatous neurosyphilis they are less favorable. In at least one clinic which has used both penicillin alone and penicillin as an adjunct to malarial fever therapy, greater success in treatment of dementia paralytica and of tabes dorsalis has been obtained with the combined method. There also are indications that penicillin therapy alone may prove inferior to combined treatment with malaria and penicillin in cases of primary atrophy of the optic nerve, late syphilitic nerve deafness and Erb's spastic paraplegia.

For the present at least, there is reason to believe that the concurrent administration of penicillin and induction of malarial fever offers the patient with late parenchymatous neurosyphilis the greatest promise of a favorable outcome. It is probably the treatment of choice, therefore, in those forms of neurosyphilis which carry a serious risk to life or important bodily function, namely, the meningoencephalitic and the tabetic form of dementia paralytica, primary atrophy of the optic nerve and nerve deafness. In cases of acute syphilitic meningitis, early or late asymptomatic neurosyphilis and meningovascular neurosyphilis, therapy with penicillin alone may be given initially, with good prospects of a favorable response.

The therapeutic problems of tabes dorsalis and Erb's spastic paraplegia require further consideration. With each, the outlook ultimately is for distressingly chronic invalidism. Since, however, the evolution of these conditions is gradual, with no immediate threat to life or vital bodily function, and since the patients frequently are in such poor general physical condition as to be poor risks for fever therapy, it is not unreasonable first to employ a form of therapy (e. g., penicillin) which is completely safe, provided there is any reasonable prospect that such therapy may be beneficial. In tabes dorsalis there is such a prospect, but in Erb's spastic paraplegia there appears to be none.

Prior to the advent of penicillin, it was believed desirable to follow malarial therapy with a prolonged course of metal chemotherapy, not only to consolidate the effects of the fever but to prevent progression of the disease in other organs, particularly the cardiovascular system. The concomitant use of penicillin may well obviate the necessity for subsequent chemotherapy and thus significantly reduce the total duration of treatment.

Moreover, with penicillin, the advantages of fever therapy have been extended to that group of patients who are unsuitable for the full course of malaria inoculations. Rose and his co-workers believe that their results with an abbreviated course of malaria inoculations, given concomitantly with penicillin therapy, were as satisfactory as those with a full course of malaria treatments alone.

Included in this editorial is a discussion of the effects of penicillin on gummatous lesions of the skin, bony skeleton and viscera. The data indicate that the healing process is no more rapid with penicillin than with metal chemotherapy. The inflammatory ocular lesions respond quickly except for interstitial keratitis, in which the results are no better than with older forms of therapy.

In the case of cardiovascular syphilis and of late latent syphilis, the evaluation of the usefulness of any therapeutic agent involves many years of post-treatment observation. There is as yet, therefore, no information as to the results of penicillin therapy in these conditions. Caution has been urged in the use of large initial doses

of penicillin in the presence of overt cardiovascular syphilis, in view of the possible complications from therapeutic shock.

It is obvious, however, that treatment with penicillin offers nothing to patients with late latent syphilis whose serologic reactions remain positive after prolonged chemotherapy. To subject these patients to further therapy of any kind solely for the purpose of attaining seronegativity is to kindle false hopes and to waste time, money and effort.

In the prevention of prenatal infection through treatment of pregnant women with syphilis, penicillin has been highly efficacious. Here it probably is, as Goodwin and Moore have suggested, the present therapy of choice. Penicillin readily passes the placental barrier, and its spirocheticidal action is available to the fetus in utero. Despite the contentions of some authors, it appears not to provoke uterine contractions or to precipitate premature labor. The outlook for a nonsyphilitic child following penicillin therapy is excellent. Even among those mothers whose syphilitic infection has been recently acquired, and in whom the risk to the child is great, there have been remarkably few treatment failures.

In treatment of syphilis, one has in penicillin a drug of negligible toxicity, readily administered but with definite limitations in therapeutic effectiveness. It is far from being the ideal therapeutic agent. Yet it has, for the present at least, a place in the treatment of syphilis as the most desirable therapeutic means at present available for certain of the protean manifestations of this disease, and as an adjunct to older methods in the treatment of others.

GUTTMAN, Philadelphia.

## Muscular System

FAMILIAL MYASTHENIA GRAVIS. LODOVICO MANCUSI-UNGARO, Ann. Int. Med. **23**:249 (Aug.) 1945.

Mancusi-Ungaro reports the cases of 2 sisters as instances of familial myasthenia gravis. In neither case was there any roentgenographic evidence to suggest mediastinal involvement. Neostigmine was effective in treatment of 1 sister, but not of the other. The author states that, as is well known, remissions are frequent in this state and that the improvement of the one sister may be attributed either to neostigmine or to the natural course of the disease. Both sisters noticed the onset of their symptoms after working in a greatly overheated room.

GUTTMAN, Philadelphia.

AMYOTONIA CONGENITA (OPPENHEIM'S DISEASE): REPORT OF CASE, WITH NECROPSY. J. A. CUNNINGHAM, South. M. J. **39**:222 (March) 1946.

The patient, a boy, seemed normal at birth but later showed flaccid extremities and coughed and gurgled as though he was having difficulty in swallowing. At the age of about 6 months he was unable to sit up or hold his head erect, and his arms and legs were flaccid. Treatment with neostigmine hydrochloride and aminoacetic acid was begun, but the infant died several weeks later. On the basis of the history and observations post mortem Cunningham stresses that in this case of clinically diagnosed amyotonia congenita (Oppenheim) the important changes were confined largely to the voluntary muscles and the central nervous system. The cause of death was bronchopneumonia, and possibly asphyxia resulting from impairment of the swallowing reflex. There were also a healing Ghon tubercle and an enlarged thymus. The muscles showed varying amounts of small embryonic muscle spindles with preserved cross striations and easily demonstrated Krause and Hensen membranes, together with some entirely normal muscle. The central nervous system showed depletion in the number of motor ganglion cells in the anterior horns of the cord and diminution in size and distortion of the remaining ganglion cells. The anterior roots were diminished in size and showed faulty or no myelination. There was no demyelination of the tracts in the spinal cord and no evidence of involvement of pyramidal cells of the cerebral cortex.

J. A. M. A.

# Society Transactions

## BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

**Madelaine R. Brown, M.D., *Presiding***

***Regular Meeting, Dec. 20, 1945***

**Extensive Bulbar Trigeminal Tractotomy: Report of Cases.** Dr. William H. Sweet, Birmingham, England.

This paper will be published in a future issue of *Brain*.

**Neuropsychiatric Services in the European Theater of Operations.** Colonel Lloyd J. Thompson, Medical Corps, Army of the United States.

Neuropsychiatric services in the European Theater of Operations were at all times part of the general medical services. From the beginning, the policy was to have a strong neuropsychiatric section in every general and station hospital. This meant neuropsychiatric wards and consultation, outpatient and rehabilitation services. Each general hospital had a specially constructed closed ward for the neuropsychiatric section.

With some caution, special hospitals for neuropsychiatric patients were organized. At first the appeal was for a unit for psychotic patients, but with increasing emphasis on prevention and rehabilitation "neurosis hospitals," or "mental rehabilitation" units, came into being. Eventually there were four special neuropsychiatric hospitals—two on the Continent and two in England. One unit in England was a holding hospital for psychotic patients, but all four carried out a program of intensive therapy. In the "neurosis centers" an atmosphere of "recovery and return to duty" was created for the patient from the moment he entered the admission office until he was dicharged. These hospitals consistently maintained an average rate of return to duty of 80 per cent, and they received patients after other units had been unable to return them to duty.

The School of Neuropsychiatry was really a part of the neuropsychiatric units. This school was opened in April 1943, under the direction of Lieut. Col. Jackson Thomas. At first neuropsychiatrists coming into the Theater with general and station hospitals were given a one month course. Along with this went courses for nurses and enlisted men. Then one week courses for medical officers of ground force units were started. In September 1943, Lieutenant Colonel Thomas was recalled to the Zone of the Interior, and it was not until late in the year that Major Howard Fabing replaced him. From that time until the middle of July 1944 the school concentrated on courses in "first aid psychiatry" for front line medical officers. Approximately 700 such officers attended, and the total attendance at the school was over 1,200.

In the Zone of Communications there were neuropsychiatric services in the two large reenforcement depots and in the disciplinary training centers. Mention should be made of the recovery center for "unwilling soldiers," which was not a medical unit but received trainees only after thorough study in a neuropsychiatric unit.

Concerning Army organization, the reestablishment of the post of division psychiatrist was recommended in September 1942, but it was not until November 1943 that this was done officially. It was necessary to train several men for these positions in the Theater. The division psychiatrist was the keystone of neuropsychiatric services in combat units. He functioned mainly at the level of the

clearing station, where he had one or two tents separate from the others for patients with combat exhaustion. An important part of his work was to see that medical officers in front of him functioned efficiently. Early in combat it was recognized that a division psychiatrist who was having a large percentage of "returns to duty" was not doing a good job forward.

All Armies except the Third Army formed "exhaustion centers" to the rear of divisions. This was accomplished by taking the neuropsychiatrists out of the evacuation hospitals and placing them in a "housekeeping" unit, such as a clearing company. The exhaustion center was in effect a small neuropsychiatric hospital where intensive treatment could be carried out for seven to ten days.

Types of cases and methods of treatment cannot be elaborated on here. It will be seen from the foregoing description that at every echelon, from the aid station at the front to the holding neuropsychiatric hospital in England, neuropsychiatric services were available for triage or adequate treatment. Within Army services, 60 per cent of the neuropsychiatric patients were returned to duty—the majority to combat duty. This does not include the patients who were returned to duty up forward at aid stations. At this rate, 40 out of every 100 neuropsychiatric patients came back to the Zone of Communications. There the neuropsychiatric hospitals returned approximately 80 per cent to duty—usually noncombat duty. This would leave only 8 or 10 of the original 100 for evacuation to the Zone of Interior, and among them would be a few psychotic patients, epileptic patients and others who could not possibly be reclaimed for any type of service.

DISCUSSION

DR. LEO ALEXANDER: It is regrettable that Colonel Thompson could not show the motion picture he had originally intended to exhibit and which he and his staff, especially Major Fabing, prepared in the European Theater of Operations. It is an excellent illustration of the methods of treatment used. The neuropsychiatric organization under Colonel Thompson in the European Theater of Operations was a very effective one. Most of its members have now returned to civilian life.

In studying and treating war neuroses, one could not help being impressed with the pavlovian mechanisms which enter into the making of the neurosis, and with the great importance which constitution plays, in addition to external stress. I found it particularly striking that, in confirmation of Pavlov's observations on animals, persons with "strong nervous systems" tended to respond with states of excitation, while persons with "weak nervous systems" tended to respond with states of inhibition, irrespective of any other diagnostic category, such as anxiety state, hysteria or reactive depression.

In patients who responded with states of excitation, deep narcotherapy of at least three days' duration followed by a period of insulin subshock treatment—combined, of course, with ventilative and encouraging psychotherapy and mobilization of the patient's own internal resources by shifting emphasis—was a form of treatment successful in many cases. Inhibited patients did better with the abreaction forms of therapy; however, in the hospital experience of my colleagues and myself none of the latter group ever attained sufficient improvement to return to combat duty, probably because in this group a greater number of basically weak personalities were present.

It is remarkable how many of the patients with states of excitation recovered sufficiently to go back to combat duty. This occurred only in those of our hospital patients who were Air Force personnel. None of our infantry personnel were returned to combat duty. This was due to the fact that whereas we served as an evacuation hospital as well as a general hospital to the Air Force, we served as a general hospital to the infantry and were thus too far removed in the chain of evacuation to receive any but its most severe psychiatric casualties.

In conclusion, I should like to say that the management of psychiatric treatment in our American Army was highly effective and was superior to that of any other

army in the field. I had an opportunity to compare our methods with the Germans', both by seeking and talking to prisoners of war and by interrogating psychiatric medical officers of all echelons in the German army after they had been taken prisoners. I recall one German medical aid man who had surrendered to our Army during the Battle of the Falaise Gap. He had been under intense combat activity, including shelling, for eighteen days. Gradually he had become tense, could no longer sleep and eat and had difficulty in forcing himself to go after wounded men under fire. He surrendered to the Americans in a setting of combat exhaustion and discouragement. When I saw him a good while later, he expressed guilt feelings about having surrendered. He described his symptoms, which I recognized as typical of combat neurosis, and I asked him whether he had discussed his symptoms with his medical officer. He replied, "Of course not. He would have had to report me, and I would have been shot."

While I found no evidence that the Germans actually shot those of their soldiers who complained of symptoms of combat exhaustion except during the final phases of the war, and then only when they were caught faking wounds by putting on large bandages, they, nevertheless, wanted to create an atmosphere in which soldiers believed that they would be shot if they complained of symptoms of combat fatigue. One of the psychiatrists whom I interrogated stated that he used that technic in his "psychotherapy."

Thus it came about, as Major Fabing so aptly worded it when I talked to him about this problem one evening in Paris, that the men with combat exhaustion in the German army tended to surrender to the enemy, while ours surrendered to their own medical officers.

MAJOR MERRILL MOORE, M. C., A. U. S.: The treatment of the psychoneuroses of war that was used in the South and the Southwest Pacific would be too complex to go into at this late hour; so I shall express only my admiration for the excellent performance of his administrative duties that Colonel Thompson has given. He is too modest to tell us much about the organization that he helped create, which amounted to more than two hundred functioning hospital units at one time. In these, so far as psychiatry was concerned, he directed all procedures, the assignment of personnel and the carrying out of policies. His administrative work in the European Theater was essentially concerned with preventive psychiatry. I hope he will turn over a complete copy of his paper to the Secretary for publication.

LIEUT. COL. WILFRED BLOOMBERG, M. C., A. U. S.: A number of things occur to me. The first has been mentioned, namely, that Colonel Thompson is extraordinarily modest. He has not given us a complete picture of organization, which was so effective in the European Theater of Operations. However, I am delighted to have the Colonel with us and it has given me a chance to hear his story with a beginning, a middle and an end.

The plans for handling psychiatric problems in the Pacific Theater followed much the pattern that was found effective in the European Theater. I think all agree that if it were possible to have strong psychiatric units in all general hospital groups it would be advantageous; but it is not possible. In spite of fancy promises and exaggerations, there were not enough psychiatrists to go around. In the Pacific Theater there was set up and functioning a large station hospital for the care of psychiatric disorders. There was set up and functioning a large general hospital for psychotic men and men who could not be returned to duty. There was set up a large general hospital for minor psychiatric disorders. In the Pacific Theater the formulation of what had been found useful in Europe was accepted.

Also lifted in principle from the European experience was the plan to move psychiatric patients out of general hospitals. If it became necessary to hospitalize them, this was done where there was a special hospital, with special personnel. When these patients had been triaged and screened at the aid station and finally got back to the Zone of the Interior, the neuropsychiatric services were able to get many back to duty.

**Kenneth J. Tillotson, M.D., *Presiding***

***Regular Meeting, Jan. 17, 1946***

**Psychosurgery: 1936-1946.** Dr. Walter Freeman and Dr. James W. Watts, Washington, D. C.

This article will appear in full in a later issue of the Archives.

DISCUSSION

Dr. James L. Poppen: I have no business to discuss this paper, considering the relatively few patients (100) my colleagues and I have operated on. In this field we have acted purely as technicians. We have depended entirely on psychiatrists at the Boston Psychopathic Hospital to choose the patients they thought suitable for this operation. Dr. Freeman and Dr. Watts should be complimented on popularizing this operation and on treating these tragic patients. When one sees the photographs of the patients before and after operation which Dr. Freeman has shown, one is inclined to laugh at the horrible faces; but it is a tragic situation, and when one meets it personally, perhaps in one's own family, it is not a laughing matter. Not only the patient suffers but the family as well. Some of these patients can be restored to a useful life. If only a few persons can be helped, it is worth while. Dr. Freeman and Dr. Watts have done a great deal to make us realize that lobotomy is of real aid and that it is under rather strong opposition in certain sections. I was rather skeptical about its value at first, but as I have followed the patients after lobotomy I have come to feel it has definite merits. As to choosing patients suitable for lobotomy, I am not qualified to speak. Dr. Solomon has made an excellent study of a series of patients before and after operation.

As to the surgical technic, it hampers me to be guided by some one else as to where to make the incision through the white matter. The open method, in which one can visualize these structures, has merit. Dr. J. G. Lyerly, of Jacksonville, Fla., suggested making a trephine opening so that if bleeding was encountered he could stop it by actual visualization. This is a constructive criticism of the closed method. It seems to me that if the surgeon can actually visualize the structures which he is dividing he can feel competent in doing what he set out to do. I have two slides to show of the method we use at the present time. The brain is somewhat like the face. No two faces are of exactly the same size or shape. The brain also varies in size and shape, especially the ventricles and convolutions. I do not see how one can be accurate by depending on measurement alone. One cannot speak about a millimeter, but one can speak about millimeters. The relationship of the anterior horn of the lateral ventricle to the sphenoid wing varies greatly, especially since many of these patients have enlarged ventricles due to atrophy.

(Slides) I wish to say something about the indications for operation which has nothing to do with psychiatric disorders, but has to do with pain. We had a patient who had severe pain in the left side of the face. A section of the fifth nerve had been performed, but she still complained. She had psychiatric treatment, was taking morphine every three hours and had lost from 180 to 100 pounds (81.6 to 45.4 Kg.) in weight. She could talk of nothing but the pain in her face. Two years ago a section of the frontothalamic tract was made. At present she is a clerk in a store, and, as I understand, is entirely satisfactory in that capacity. She goes to card parties, is perfectly happy and has never asked for morphine or for any other drug since the operation. Dr. Freeman stated that he had a patient on whom a lobotomy was performed for relief of pain associated with carcinoma. Lobotomy is certainly a potentially less serious operation than chordotomy. In some cases lobotomy is of real value in the relief of pain from a fatal carcinoma.

I have been somewhat hesitant about lobotomies in young girls. I wonder about the effect on their morality. Can Dr. Freeman and Dr. Solomon tell us something about that?

DR. WILLIAM JASON MIXTER: I have been interested in this work ever since Dr. Watts and Dr. Freeman started it. It seemed to me that it was a rational procedure, to be carried out in selected cases. It is interesting to see this series of cases enlarged. Dr. Solomon has said there are thousands of patients in Massachusetts who should be operated on. My associates and I have followed the technic of Dr. Freeman and Dr. Watts rather closely at McLean Hospital and at Massachusetts General Hospital, and so far we have been pretty well satisfied. We have had 1 fatality, presumably because the section was placed too far back. It seems to me that we who are surgeons must be guided by the psychiatrists concerning indications for operation; it is their field, not ours. We started with a very small group of patients with agitated depressions. We have operated on 3 patients with pain, 1 of whom was the patient who died. The other 2 were operated on too recently for me to be able to report results, but they seem to be definitely better. I have always questioned a bit the value of lobotomy for schizophrenic patients, and I shall be interested to see how they respond.

It has been a great pleasure to hear Dr. Freeman again, and I only wish that Dr. Watts were here too, to give us his opinion about the surgical approach to this interesting problem.

DR. HARRY C. SOLOMON: As a result of Dr. Freeman and Dr. Watts's pioneering for use against rather widespread skepticism, a noteworthy contribution to psychiatry has been produced. For a number of years many of us read the reports of Drs. Freeman and Watts and thought that, next to Ananias, they were the best story tellers and the best liars the world had heard of. It was argued that it stood to reason that people could not get well because a piece of brain was removed. I held aloof until two years ago, when, in a moment of irresponsibility, I was pushed into this work. Now, like all converts, I should like to do a little evangelical work. The results which my colleagues and I have had have amazed us. Dr. Freeman's statistics are much more valid than ours because of his longer time of observation. At first we thought that this radical operation should be done only on patients with chronic disease of several years' duration which every one had put themselves on record as incurable. Most of these patients had had electric shock, and many of them insulin shock, to see whether they would recover. They were patients who had had from two to twenty years of hospitalization. They made up the majority of our group, and we went rather slowly; but we have had extraordinary results. Up to August, our series contained 23 patients with chronic schizophrenia—very sick patients. The state hospitals sent us patients who presented the most distressing problems in handling. By November 8, 8 of these patients had gone home, 3 were about to be discharged, 5 were front ward instead of back ward patients and a few did not show good results. Some of our patients have made a really extraordinary recovery or improvement. A Harvard graduate, who had been five years in the hospital, is now doing exacting work. The most striking result is a warmth of feeling, i. e., warmth in the expression of affection to their families, which these patients generally show. To see these vituperative and scolding women on admission and then three or four days after operation to see them greeting their families with kind and affectionate regard is an amazing experience. The families are thrilled. You can imagine what they say to us. It is a heartening situation.

That the patients with agitated depressions should show a release of their agitation, quiet down and become rather productive citizens is one thing, but that these chronic, hopeless, unquestionably schizophrenic patients should take a new lease on life and become reasonable persons again is something that has rather shaken my stability. One should give a great deal of credit to Dr. Freeman and Dr. Watts for opening this avenue of endeavor. I am sure there are hundreds of patients' relatives in this country who look on them with something more than kind regard.

Dr. Freeman says patients tend to lose the sense of forecasting the future. I do not think that this is true. I should like to reassure Dr. Poppen's New England

conscience, which makes him worry about the morality of the virgins. Actually, our patients have more, rather than less, of the moral virtues after lobotomy. They have a greater degree of good morals than they had just prior to and during their psychotic behavior. We have been impressed with the fact that, contrary to what one might conclude from the literature, we have not produced vegetative, adipose, lazy, overeating patients. Many remain rather slim and sleek and have better figures than before the operation. I wonder whether a difference in the technic of operation may account for a difference in results.

I suppose all neurosurgeons who perform this operation do it differently, and therefore it has a different effect on the brain. From the mechanistic standpoint, the results should differ according to the place at which the cut is made. We like to think that with the skilful surgery of Dr. Poppen and Dr. Horrax some of the difference in results is due to differences in technic. I shall, finally, call attention to the fact that these patients have a great many other changes in their body responses. There are changes in controls of the autonomic nervous system. Perhaps Dr. Rinkel will tell us something about these effects. Many of the patients with hypertensive disorders have a return to low blood pressure level after operation. There are many extraordinary changes in the physiologic activity of the patient. Our first patient to be operated on had had diabetes for eight years and has been cured of the disease.

DR. MAX RINKEL: Bilateral frontal lobotomy offers a great opportunity for neurophysiologic studies. Freeman and Watts, in their remarkable book on "Psychosurgery" (Springfield, Ill., Charles C Thomas, Publisher, 1942), devote a chapter to the "Relation of the Frontal Lobe to the Autonomic Nervous System." With Drs. M. Greenblatt, G. P. Coon and H. C. Solomon, at the Boston Psychopathic Hospital, I have undertaken a systematic study of the autonomic nervous system on patients who had had bilateral frontal lobotomies. The sympathetic nervous system was examined after intravenous injection of 0.05 mg. of epinephrine hydrochloride in 1 cc. of sterile water over a period of ten seconds. The blood pressure, pulse rate, pulse pressure and other clinical reactions were recorded. The systolic blood pressure rose to twice the level of the controls, and the pulse pressure was tripled. Clinically, shivering and pilomotor reactions were produced. The parasympathetic nervous system was examined by stimulation of the carotid sinus. The effect of stimulation of the carotid sinus was recorded simultaneously with the electroencephalographic and electrocardiographic records. The electroencephalogram in over 90 per cent of cases was characterized by the appearance of high voltage slow waves in the 2 to 5 per cent range of frequency. The electrocardiogram in over 80 per cent of cases disclosed increased QRS-QRS intervals and heart block. Tonic-clonic convulsions occurred in almost 90 per cent. Atropine, in an amount sufficient to produce vagus block, and amphetamine sulfate abolish the carotid sinus reflex, whereas mecholyl enhances it.

Illustrative lantern slides were shown.

DR. KENNETH J. TILLOTSON, Belmont, Mass.: I should like to make a few comments in behalf of my colleagues on the staff of McLean Hospital. We have performed this operation in two series of patients. The first series (1940) consisted of 5 patients with severe agitated depressions. Three made excellent recoveries and 2 had effective changes. In the second series were 11 patients who had been schizophrenic from ten to thirty years. Obviously, we should not expect any remarkable result from patients as deteriorated as these. However, some have shown improvement in their conduct. This raises an important psychologic problem. Some types of schizophrenic patients should have this operation earlier. The early history could show that the prognosis was hopeless. With an early diagnosis, why should not prefrontal lobotomy be done early? The electric shock therapies are not very successful in cases of severe obsessional tension states. Dr. Freeman and Dr. Watts have shown that some schizophrenic patients have gone into the Army after successful recovery from lobotomy.

The cerebral disturbance occasioned by this operation should be considered. One does not know what takes place as a result of the operation or what is the nature of the effect of years of mental illness, an effect which is called deterioration. There is a great deal of work to be done. Many of the symptoms can be reversed with therapy. After lobotomy the "total push" program should add to the beneficial results. A program of reeducation is important in follow-up treatment. The importance of operation on selected schizophrenic patients as soon as the diagnosis can be made is to be emphasized. Certainly, in cases of persistent obsessional tensions it should be done.

DR. FREDERICK WYATT, Belmont, Mass.: The problem of prefrontal lobotomy as a therapeutic measure is one of personality. The way to study the changes which the personality undergoes after operation is through general psychiatric observation and the application of standardized tests. What is known about differences in personality before and after operation? I should like to ask Dr. Freeman about recent advances in psychologic studies, such as were surely undertaken in continuation of those reported in "Psychosurgery." It will be recalled that Dr. Thelma Hunt made the psychologic studies which appeared in that book. She tested 40 patients before and after operation. These patients were selected on the basis of their willingness and ability to go through this procedure. This very fact suggests, however, that the patients were accessible to communication and capable of relatively organized responses and that they were, if at all, not more than slightly deteriorated. Does not this raise a problem? Let it be assumed for present purposes that there are three states of personality: (1) the premorbid personality, (2) the mentally ill personality (if this illness is a schizophrenic psychosis it may have led to deterioration) and (3) the postoperative personality. Dr. Hunt's observation that the intellectual abilities of the patients, as reflected in her tests, were not impaired by the operation is as valid as it can be under the given conditions. One does not know as yet what changes actually occur. Patients with obsessional neuroses are known to maintain a relatively high intellectual acumen, even with the most disabling symptoms. One must not be surprised when the patient shows no major changes in personality. The very fact that psychologic studies were limited to cooperative patients tends to skew the results. On the other hand, there are indications that frontal lobotomy does bring about intellectual changes. Dr. Hunt may not have pressed the point far enough. One would wonder especially whether the ability to perform abstractions and generalizations in Goldstein's sense—a complex of functions which seem to be identical with higher intellectual abilities—had not been impaired by lobotomy and would want this aspect scrutinized with special care.

A few words about our experience with lobotomy at McLean Hospital: The problem there is the reverse of the one just discussed. More than 1 of our patients subjected to lobotomy had been ill for over thirty years and was inaccessible. We were limited entirely to postoperative observations. But lobotomy, in addition to its curative prospects, is an extraordinary experiment—if one applies it, one must make full use of it. The next problem is how to lift a chronic patient out of his state long enough to test him. Various drugs may prove helpful. If one could make systematic studies before and after the operation on patients who were chronically and severely disturbed, one might not only know more definitely what changes of personality lobotomy effects but might also learn more about the obscure concept of deterioration. Every opportunity for postoperative study should be utilized. Observations by the nurses while the patient is in the ward offer a fruitful opportunity. Another is supplied through occupational therapy. My colleagues and I have been working on a form recording the more important and revealing behavioral manifestations of the patient from day to day in that activity. We shall attempt to integrate psychiatric and psychologic data, observations of the nurses and occupational therapy records in order to obtain as comprehensive and multidimensional a picture as possible.

DR. KENNETH J. TILLOTSON, Belmont, Mass.: I should like to ask Dr. Freeman several questions: In patients with obsessional tension states and in patients with the early stages of schizophrenia who have not shown any striking deterioration after prefrontal lobotomy, what type of loss of function, if any, have you observed? Does this operation tend to produce invalidism or deterioration in the patient? The symptoms of "silliness" are, I think, due to the long years of illness preceding operation. Does the electroencephalogram change, and do you routinely inject iodized oil and take encephalograms? You mentioned home environment after operation. What sort of occupational environment do you recommend if patients remain in the hospital?

DR. WALTER FREEMAN, Washington, D. C.: Your discussers have dealt kindly with me. I recall vividly an occasion on which I appeared at a meeting of the Chicago Neurological Society nine years ago; many disparaging remarks were made, and one person said the operation was criminal. I think the attitude has changed because the proof of the pudding is in the eating. I believe it will be of value to answer questions first and then, perhaps, discuss more fully a few of the conclusions which Dr. Watts and I have reached.

What measure of loss of function is there in these cases? What defects in intelligence should be? It is largely a matter of what one calls intelligence. Webster defines intelligence as the ability to apply new methods in the solution of new problems. The problem that confronts most of our patients is that of getting along in the outside world. Take the patients who are suffering from obsessional tension states: Whereas 17 per cent of a fairly large series of such patients were remuneratively employed prior to operation, the figure rose to 67 per cent after operation. These persons are free, cheerful, relaxed and able to carry on a conversation on a variety of topics. They are alert and interested in the happenings of the day. Their capacity for carrying out the various tests, I think, is limited by their satisfaction with their ability to give some answer, not necessarily a perfect or an all-inclusive one. This is the one phenomenon which shows up in a number of the so-called tests of personality function in the Rorschach, the mosaic and the personality test and, to some extent, in the categorical aptitude test, the maze test and the various sorting tests. There probably is a permanent reduction in the richness of the associations which enable a person to see all the implications of the questions and of his replies. In other words, the patient after lobotomy seems to finish his thinking more quickly and with greater satisfaction to himself than the normal person. That is the reverse of the patient who falls into the neurotic or obsessional tension state, who is never satisfied with the results of his thinking.

Electroencephalographic studies have been carried out on our material by Dr. Robert Cohn. They showed that in the early stages after operation there was a disorganization of the alpha rhythm in the frontal leads. There was no particular correlation with the clinical result. Some patients had abnormal records before operation with later improvement. However, the electroencephalograms of others were normal, even though there was pronounced deterioration and repeated severe convulsive seizures had occurred.

Roentgenograms are taken because we want to know why operation sometimes fails. If we find from study of the roentgenograms that the incision has been made with the plane too far forward we are obliged to carry out a second operation to correct that. If the plane is too far posterior, apathy, inertia and permanent invalidism result.

We believe in getting the patient home soon after operation. He passes through a dull stage and then becomes childish, exuberant and overactive, a condition that one recognizes as normal in a child. This phenomenon should be emphasized to the relatives. They should be informed that the patient will misbehave. He may not, and so much the better; but if he does relatives will then not blame the surgeon too much. It is worth while to point out that before operation the patient was not only behaving pretty badly, so much so that he had to be locked up, but was

unhappy and suffering. After operation the behavior is probably no worse; but the patient is not suffering and the family finds it difficult to pity him and treat him like a sick person. When it is explained that he is still sick, but in a different way, and still needs guidance, the family is likely to be more patient.

We have had patients who returned to work seven or eight years after operation. Before operation they had been disabled ten or twelve years. I believe that management of patients in the hospital requires total push, as Dr. Tillotson emphasized We believe the patient should go home early because that constant social pressure brings him back sooner to adequate social behavior.

I wish I could answer Dr. Wyatt more adequately in regard to intelligence testing. During the war it was impossible to obtain adequate psychiatric studies of these patients. Dr. Wyatt's criticisms are well taken. We hope to correct the shortcomings to some extent in the future. He has made some interesting comments on the deterioration in relation to these tests. It is wholly futile to base one's conclusions concerning intellectual capacity on results with deteriorated patients. One must choose the best patients, patients who maintain a vigorous intellectual life and are disabled by obsessions that do not lead to dissociation of personality. I should choose the patients presenting anxiety states, obsessive tension states and other conditions in which the dissociation of personality is at a minimum.

With respect to surgical technic, I do not think one can say this procedure is better than that until a number of patients have been followed over a period of years. We prefer to place the incisions where we feel they should be. We like to draw on the roentgenogram the place where the incision should lie. That will depend on our estimate of the patient's age, intellectual attainments, social status, psychotic condition and deterioration. We cannot expect to get an adequate result in a patient with chronic schizophrenia by placing the incision in the plane of the coronal suture. We aim to place it 6 to 8 mm. behind it. If we are 3 mm. off in either direction, we may get a bad result. I watched Dr. Poppen do two and a half lobotomy operations today, and I must say they went through with neatness and dispatch and with little bleeding. I could see what he was doing. I could see the bulge of white matter and the various stages of operation. I am pretty sure I could see the anterior cerebral artery in 1 case. My criticisms of Dr. Poppen's method is that he has no permanent record of the exact plane of the incision. He could use tantalum powder and take roentgenograms. We use iodized oil almost routinely. It stays in the incisions. There is no reaction, and we have not noticed any ill effects from its use. Before a final estimate of the value of the open versus the closed operation is made, we shall have to wait a number of years and calculate the results. Will Dr. Poppen get 50 or 20 per cent of his patients back to work? Will he get 50 or 20 per cent out of the hospital and back to their home environment? How many of his patients will relapse; how many will have seizures, and how many will die during operation? I think the Poppen and Lyerly technics will yield adequate results.

Dr. Solomon stressed the ability of these patients to live in harmony with themselves and their environment and to adjust themselves to the needs of the moment. I agree they do handle themselves well. They can go out in the world and attend to the needs of themselves and their customers. Lack of foresight occurs in the early phases and later, particularly in the field of interpersonal relationships. If they would count 20 before they speak and 50 before they strike, it would be better. Many of them have an abundance of energy no longer trammeled. They finish their thinking more quickly than the average person. They are direct, practical and uninspired. It takes some indirection, impracticality and inspiration to produce schizophrenia, agitated depression or an obsessive tension state.

Disturbances in morality have been minor. Some patients have been picked up by the police for indecent exposure, but only because of an imperious need to urinate. Fortunately, in our series there have been no serious moral deviations.

Three of our patients married and have had children. I watched 1 of these women through her delivery, and this observation has some relation to the question of pain. During the labor pains she screwed up her face and tugged and sweated and breathed hard, like any one. Between pains she was cheeful, smiling, alert and exhilirated. There was no sham connected with her behavior.

The more this operative procedure is being used, the more indications there are for it: first, for the agitated depressions; then schizophrenia; then the obsessive tension states, for which it is truly specific, and then hysteria, particularly when associated with a great deal of pain. We had a patient, a woman, aged 40, who had gone through twelve or eighteen abdominal operations. She had sustained a minor blow on the head and had had a subtemporal decompression. She also had severe contractures of the limbs with ankylosis and had spent two years in bed because of pain. On the second postoperative day she turned over on her back with a few winces, but these subsided. On the third day she straightened out her legs. On the fourth day she walked. The pain was severe, but she was no longer afraid of being hurt. The operation was done more than nine years ago, and she has been keeping house and has been regularly employed for the last seven years.

Dr. Rinkel has shown us some interesting work, and Dr. Solomon has presented interesting facts regarding diabetes. We operated on a woman with hypertension in 1937. At that time she had had hypertension and agitated depression for eighteen years, with a blood pressure of 240 systolic and 120 diastolic. When I saw her nearly nine years later, her systolic pressure was 160 and she was leading a contented life in California. Another patient, who had schizophrenia, also had severe rheumatic carditis, and auricular fibrillation. This condition had resisted treatment with nauseating doses of digitalis; after operation the rhythm became normal. Later she had a mental relapse and died of congestive heart failure. Another patient, with psychoneurosis, had had two years of psychoanalysis. He also had subacute bacterial endocarditis. Three operations finally relieved him of his obsessive preoccupation. The endocarditis also cleared up, and the electrocardiogram became normal. Dr. W. W. Eldrige, chief medical officer at St. Elizabeths Hospital, told me of a tuberculous patient who needed five attendants to give him injections. Two operations were required to abolish this behavior, but he gained 40 pounds (18.1 Kg.) and there has been progressive clearing of the pulmonary picture. In the back wards there was a patient who had had to be fed with a tube twice a day for eleven years. It took three men to hold him and one to pass the tube. That was two years ago. He was referred for prefrontal lobotomy. He continued to require tube feedings; one day, however, the attendant went away and left the tube by the side of the patient, and the patient passed the tube himself. A few months later he used only the funnel, and he got a full meal of spaghetti. Now he is feeding himself with a spoon. Why is the recovery progressive in stages this way over long periods? I think the answer to this question contains the nub of the problem of the effect of prefrontal lobotomy. The patient's deviated behavior is the result of cerebral activity, and I believe one must postulate an anatomic substrate for organized, stereotyped behavior. In lobotomy the cortex is traumatized to only a limited degree. The most significant part of the operation is the severing of connections of the frontal lobe with the rest of the brain, particularly the connection between the frontal lobe and the thalamus. In my opinion, it is the emotional component of the psychosis that is removed, abolished, destroyed by the operative procedure, and it is reasonable to assume that it is the emotional component which keeps the pychosis alive. When that emotional component is removed, there is no longer any good reason for the continuation of this peculiar behavior. It has less meaning for the patient; the stimulus is no longer relevant. Therefore the behavior which is carried out after operation undergoes a gradual resolution, with disappearance of most of the abnormal forms.

## ILLINOIS PSYCHIATRIC SOCIETY

**John J. Madden, M.D., *President, in the Chair***

***April 4, 1946***

**Memorial: Charles F. Read, M.D.** DR. GEORGE A. WILTRAKIS.

**The Hostile Dependent Behavior Factor in Rehabilitation of Veterans with Psychoneuroses and of the Industrially Injured with Psychologically Protracted Convalescence.** DR. ALFRED P. SOLOMON.

One of the most rejected men in society is the industrially injured workman who has a protracted convalescence for psychologic reasons. These are men who have been determined by the surgeons as able to return to work; yet their behavior is a hostile denial of this opinion. This hostile dependent behavior is confronted not only with the traditional contempt of society for those who do not accept responsibilities but also with the contempt of the employer milieu and family. An even severer critic is his own conscience, which does not permit him to face his dependent attitude but, instead, forces him to behave as a sick man needing medical care. The paradox is that, though his complaints are many, what he seeks is not treatment but a diagnosis that will appease his conscience.

The state compensation laws and the general lack of understanding of the psychologic factors involved are largely responsible for the existence of this behavior.

At the state-operated Veterans' Rehabilitation Center of Chicago, experience has been gained in the rehabilitation of veterans with psychoneuroses which can be utilized in the treatment of the industrially injured with a psychologically protracted convalescence. The treatment at the center has as its basis an understanding of the psychologic factors in human relationships. It utilizes an all-day therapy involving recreational and physical educational activities, the arts and crafts, socialization activities and individual and group psychotherapy, all prescribed by the psychiatrist to fit the individual needs of the veteran, so that the rehabilitated veteran will better be able to face the vicissitudes of life and avoid future breakdowns. This vocational rehabilitation program is recommended on a basis of study by a staff psychologist and social worker, as well as a psychiatrist. Important in the treatment at the center is an integration of the teamwork, psychologically oriented, among all members of the staff. The advantage of treatment at a state-operated rehabilitation center is that there is responsibility only to the veteran, the matter of adjustment of pensions requiring no consideration by the staff at the center.

It is believed that the psychotherapeutic lessons learned at the center offer an answer to the parallel problem in industry presented by the injured workmen with psychologically protracted convalescence. However, before psychiatry can make use of these new psychologic technics in rehabilitation, state compensation laws must be investigated by a commission composed of psychiatrists, industrial surgeons, labor leaders and insurance men, so that revision can be made to utilize the pychiatrist as a therapeutist rather than as a medicolegal diagnostician.

**Psychiatric Aspects of Shoplifting.** DR. ALEX J. ARIEFF and DR. CAROL G. BOWIE.

In a study of 338 shoplifters referred by the municipal courts to the Psychiatric Institute of the Municipal Courts of Chicago over a five year period, the following facts were elicited:

Between 25 and 30 per cent of arrested shoplifters were referred for psychiatric examination by the courts. Arrested shoplifters constituted about 5 per cent of all apprehended shoplifters.

The value of the articles taken by this group was usually small, averaging $20 in cost and being of little value to the patient.

There was more shoplifting in the spring and just before Christmas.

The majority of patients were women; 313 of 338 patients analyzed were women.

In 1942 the ratio of these offenses to the total case load was highest, whereas in the last three years of the war the percentage dropped appreciably.

The age groups most involved were the early immature and the middle-aged. Many women in the latter were in the menopausal period.

Of 338 patients, 77 per cent had a definite mental or emotional disorder. The diagnosis for a large percentage of patients was acute anxiety state with mental depression. Marital discord, low economic level and low intellectual level were not factors.

Even the war's continuance did not increase the incidence of shoplifting.

The majority had no previous arrests. Of 42 with previous arrests in 100 sample cases, 24 per cent had had arrests for the same offense.

Forty-nine per cent of the patients were disposed of by court supervision, 36 per cent by psychiatric supervision, 5 per cent by correctional care and 12 per cent by commitment.

# News and Comment

## TRAINING PROGRAM FOR PSYCHIATRISTS

The American Board of Psychiatry and Neurology, Inc., makes the following suggestions for a three year full time training program for psychiatrists:

1. One year of inpatient work with an adequate variety of psychiatric conditions.
2. Six months of full time outpatient clinic work, or its equivalent, with emphasis on the study and treatment of psychoneurotic patients, with a minimum of twenty interviews per week per resident.
3. Six months of work in neurology—half-time clinical and half-time basic.
4. Six months of half-time service in the psychiatric aspects of general medical and surgical conditions.
5. Six months of half-time work in child psychiatry and experience in working with psychologists and psychiatric social workers.
6. Six months of specialized institutional psychiatry (feeblemindedness, epilepsy, forensic psychiatry, penology, drug and alcohol addiction, and so forth).
7. During these three years it is recommended that there be available teaching ward rounds, staff conferences, seminars, journal clubs, adequate psychiatric texts and periodicals and participation in some phase of psychiatric investigation.
8. During these three years there should be adequate instruction in the basic psychiatric concepts, as covered in the material recommended in the syllabus of the American Board of Psychiatry and Neurology, Inc.
9. In institutions in which there is no full time senior staff there should be given in the aggregate a minimum of fifteen hours a week of service by the senior attending staff in capacities instructive to the resident staff.
10. In planning or evaluating training, one, two or three year programs may be worked out to include various fractions of the foregoing suggested items. For instance, a resident may devote a full day or a half-day a week to the psychiatric aspects of medical and surgical conditions for a year or so while assuming major clinical responsibilities in a psychiatric hospital.

The only purpose in suggesting the foregoing program is to indicate a desirable spread of experience in the training of a psychiatrist. It is thought unwise for any teaching program to be rigidly or slavishly followed.

## RESIDENCY IN PSYCHIATRY, VETERANS ADMINISTRATION HOSPITAL IN NEW ORLEANS

The Veterans Administration Hospital in New Orleans offers a two year residency in neuropsychiatry. This includes work with all phases of the psychoses and psychoneuroses as well as the usual types of neurologic and neurosurgical cases commonly encountered in a five hundred bed hospital. The hospital has recently been designated as a neurosurgery center, and this will increase the number of cases available for neurosurgical study.

In addition to the hospital work, the residents are detached from their duties for a period of six weeks each year, during which they are enrolled as graduate students in the departments of neuroanatomy and neurophysiology at the Tulane University of Louisiana School of Medicine. The department of neuropsychiatry is staffed by members of the faculty of the school of medicine of Tulane University who work at the hospital on a part time basis as consulting and attending physicians. In addition, a professor of psychology and several graduate students study the patients from the various psychologic angles. A full time social service worker and several graduate students study the patients from the social standpoint.

For information write the chairman of the dean's committee, Tulane University of Louisiana, 1430 Tulane Avenue, New Orleans 13.

## KANSAS PSYCHIATRIC SOCIETY

At a recent meeting of the Kansas Psychiatric Society the following officers were elected for the ensuing year: Dr. Lewis L. Robbins, president; Dr. J. F. Casey, vice president; Dr. Edward D. Greenwood, secretary-treasurer, and Dr. E. M. Wiedenmann and Dr. Paul E. Davis, counsellors.

# Book Reviews

**22 Cells in Nuremberg.** By Douglas M. Kelley, M.D. Price, $3. Pp. 245. New York: Greenberg, Publisher, Inc., 1947.

This interesting book introduces no new facts about the twenty-two war criminals Many descriptions fall short and do not give a clear pattern of their personalities. Not new, but significant, is the statement that the trend to aggression has been present in the German people for many decades.

Of all the men on trial, only one, Robert Ley, had pathologic changes in the frontal lobe of the brain. Hitler and Hess showed psychopathologic pictures. Both suffered from hysterical reactions with disturbances of reality and paranoid features. All the others were men with ambition, ability and some cruelty. They were much the same as the people one meets in daily life in offices as business executives, union leaders, officials and others. There was nothing extraordinary about them. The accused and the German people had one thing in common—a lack of maturity, which in the case of the German people was exhibited in the emotional field.

The author answers the argument of the Americans that "we are much too civilized for such a situation to develop and could never regress to a point where a small group could seize total power." He states, "Shocking as it may seem to some of us, we as a people greatly resemble the Germans of two decades ago." He charges the American people with the same immaturity as the Germans. The author feels that the heterogeneity of the structure of the American people produces various forms and states of immaturity. This is a weak protection against a dictatorship by a single man or a small group of men. He feels that only maturity, achieved by education, is a safeguard against events such as those which took place in Germany. It is a remarkable book, which can be highly recommended.

**Textbook for Psychiatric Attendants.** By Laura W. Fitzsimmons, R.N. Price, $3.50. Pp. 332. New York: The Macmillan Company, 1947.

In many psychiatric hospitals, the actual daily care of patients is given by attendants. Capable, well trained and enthusiastic attendants contribute in an important way to an efficient psychiatric service.

As indicated in the foreword of this volume, the Nursing Committee of the American Psychiatric Association has focused on the problem of adequate and standardized training for the attendant. With the support of that committee in 1945, Laura W. Fitzsimmons first published "A Manual for Training Attendants in Mental Hospitals," giving a basic formula for a teaching plan.

The volume at hand is a text that may be used by the attendants taking the course outlined in the manual or by those who do not have the benefit of classes.

In reading this book one is struck by the wealth of practical material given in a concise and usable form. While there is sufficient material of a more theoretic nature, in accordance with the modern tendency in teaching nurses and attendants, emphasis is on symptoms rather than diagnoses. Among the topics covered are: the attitude of the attendant, admission procedures, ward observations, suicide prevention, restraints, the handling of visitors, emergencies and first aid, ward housekeeping, mental hygiene for attendants, the attendant's place in occupational therapy, hydrotherapy, the care of disturbed patients, the details of flow baths, packs, administration of medicines and gavage. A section on electric shock and insulin therapy might have been included. While it would be ideal if nurses were always available to assist with these treatments, in actuality in some hospitals the shortage of nurses necessitates the use of attendants.

This volume should be recommended to attendants and will be useful to psychiatrists and nurses concerned with the instruction of attendants.

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